PUGET SOUND
Clean Air Agency

Puget Sound Air Toxics Evaluation

May 2002

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Executive Summary

The Puget Sound Clean Air Agency conducted this study in an effort to better understand the potential health risks to our three million residents from a group of air contaminants commonly referred to as air toxics. This study is intended to assist the agency in improving its air toxics regulations and voluntary programs and to provide more information for allocating resources. The estimates of cancer and non-cancer health effects should not be viewed as actual cancer or non-cancer cases resulting from air pollution but as an estimate of relative impact of the evaluated toxic-air pollutants so the agency can prioritize its efforts to reduce exposures.

Defining Air Toxics

Air toxics are different from the six traditional air pollutants or "criteria pollutants" that have been regulated by environmental regulatory agencies for a number of years. Our agency defines "air toxics" as a broad category of chemicals that covers over 400 air pollutants along with woodsmoke and diesel particles. Similarly, the United States Environmental Protection Agency (U.S. Environmental Protection Agency) commonly refers to "air toxics" as a synonym for the 188 hazardous air pollutants listed in the 1990 amendments to the federal Clean Air Act. Because resources are not available to evaluate every chemical, this study evaluates a short list of 18 to 32 air toxics. We hope to expand the list of toxics when more resources become available.

Methods

This study uses basic risk assessment concepts and models, such as toxicity and exposure assessment, to provide a general overview of the potential health impacts that could be due to air toxics. Because of limited resources, this report does not perform a comprehensive risk assessment, which would include more detailed analyses and discussion of toxicity and exposure parameters, as well as a more in-depth risk characterization section. More comprehensive information on various details of this study can be found in the technical support documents which are referenced throughout this report.

Toxicity

The toxicity chapter includes dose-response information on the variety of air toxics evaluated in the Puget Sound region. The majority of this information is based on toxicity analyses performed by USEPA and included in their Integrated Risk Information System (IRIS). For some chemicals and mixtures, such as diesel particulate matter, chromium, and woodsmoke, we depart from recommended USEPA IRIS toxicity values. For example, for diesel particulate matter, we use the California Environmental Protection Agency's toxicity evaluation. Our rationale for this and other departures are described in the toxicity chapter.

Exposure

The toxicity values described above are combined with exposure assessment information to estimate both cancer and non-cancer potential health risks. We use results from three different exposure assessments to characterize air emissions and to estimate potential exposure concentrations for the residents of the Puget Sound area. These three exposure assessments include a monitoring study conducted in the greater Seattle/King County area, and two modeling assessments conducted as part of USEPA National-scale Air Toxics Assessment (NATA) in the four counties in the Puget Sound Clean Air Agency jurisdiction (King, Kitsap, Pierce and Snohomish counties).

The monitoring study, which was conducted by Washington State Department of Ecology in partnership with the Puget Sound Clean Air Agency and USEPA, sampled outdoor air at six different locations throughout the greater Seattle/King County area during 2000 and 2001. These six locations include areas near or in Beacon Hill, Georgetown, Lake Sammamish, Lake Forest Park, the Maple Leaf reservoir in north Seattle, and the City of SeaTac.

In addition to the monitoring study, we used exposure estimates from two models used by USEPA in their nationwide air toxics study entitled the National-scale Air Toxics Assessment (NATA). In this study, USEPA predicts outdoor air concentrations using the

ASPEN model for 32 air toxics in counties across the country. We obtained the outdoor air concentrations for the four Puget Sound counties, compared them to monitored concentrations, and calculated potential health risks associated with those concentrations.

The third model used to predict exposure concentrations is also part of the NATA study. This model, entitled the Hazardous Air Pollutant Exposure Model, predicts human exposures to the outdoor air pollutants by considering typical human behaviors and environments where these outdoor pollutants might accumulate. For example, this model uses average commute time estimates for a variety of individuals to estimate potential exposures to vehicle exhaust while riding in cars or waiting in traffic. Exposures such as these are combined for multiple activities and locations to estimate an average exposure concentration for each of the 32 air toxics.

All exposure concentrations are based on annual averages or medians (the 50^{th} percentile), and residents are assumed to be exposed for 70 years, an average lifetime for an individual. We also assumed that these residents are healthy adults. We did not include exposure or toxicity adjustments specific to children. Some health-protective assumptions are included in the toxicity estimates that may protect sensitive people such as the elderly or diseased individuals.

The health risk estimates are based on a combination of average and reasonably conservative or health-protective assumptions. *This is expected to lead to risk estimates that are reasonably high for the chemicals included in the analysis, but not worst case*.

Results

The primary health effect of concern from the chemicals evaluated in this study is cancer. More specifically, lung cancer is associated with both diesel soot and woodsmoke, although it is also associated with 1,3 butadiene, a mobile source related contaminant. In addition to lung cancer, leukemia, nasal and liver cancers are associated with chemicals that ranked high in our study. The majority of the cancer risk estimated in our study is

due to diesel soot. On average, diesel soot accounts for somewhere between 70 to 85 percent of the total cancer risk from air toxics in our area.

Our study found that the significant non-cancer health effects from air toxics in our area are primarily due to acrolein. This chemical is associated with upper respiratory irritation.

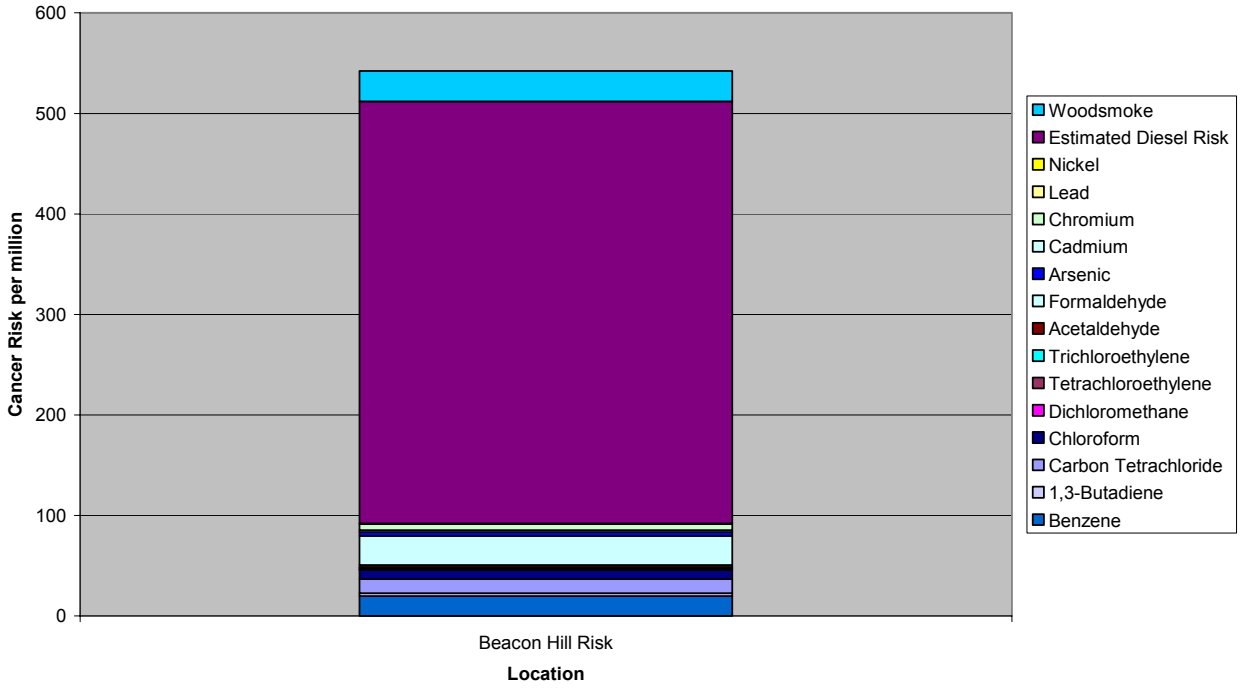
It is important to note, however, that our study does not include the serious non-cancer health effects associated with the particle fraction of two air toxics, diesel soot and woodsmoke. Non-cancer health effects associated with these particles have been extensively studied and documented in the scientific literature, and a full analysis is beyond the scope of this study.

Potential Cancer Risks

The average cancer risk estimates, even when human and pollutant movement are considered, are similar among the different methods of calculating exposures concentrations, and across different areas of the Puget Sound region. For example, average cancer risk estimates for King County alone range from approximately 400 to 700 in a million based on 32 air toxics from the human exposure model and outdoor model data, respectively.

The average cancer risk estimates for the monitored data are approximately 550 in a million for the greater Seattle/King County area (see Figure ES-1). As described above, the monitoring study only looked at a total of 17 air toxics. The total cancer risks associated with the King County modeled estimates are higher because it includes more chemicals, not because the estimates of each chemical are higher.

Figure ES-1: Potential Cancer Risks Estimated for Beacon Hill (2000) Monitoring Data (17 air toxics)

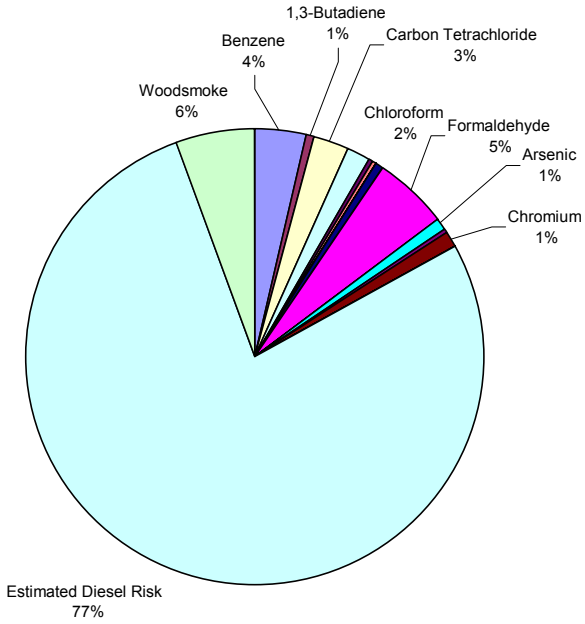


The average cancer risk estimates are also similar in the remaining three counties in the Puget Sound jurisdiction (Kitsap, Pierce, and Snohomish counties), although we do not have monitored information to confirm our findings. The estimated cancer risks range from a median value of 300 in one million for all 32 air toxics human exposure modeled toxics, including diesel soot, in Snohomish County to a high of 690 in one million as an average for 32 modeled ambient concentrations in King County, including diesel soot. All risk estimates reflect a 70-year exposure period.

The air toxics that contribute most to the cancer risks are also consistent across the different methods of analysis. **The top toxics for all three methods include diesel soot, benzene, formaldehyde, and carbon tetrachloride**. **Woodsmoke** also contributes significantly to the risk estimates based on the monitored data.

In addition, the percent contribution of the top air toxics is also very similar across the different methods of analysis. For example, at Beacon Hill, diesel soot accounts for over 75 percent of the potential cancer risks with woodsmoke contributing approximately 6 percent (see Figure ES-2). The King County results from the outdoor NATA model estimate diesel particulate matter at 86 percent, with other mobile-source-related chemicals at about 8 percent, and stationary-source-related chemicals at about 6 percent. Similarly, the NATA human exposure results indicate a diesel soot contribution of 86 percent, with other mobile-source-related chemicals at 7 percent, and stationary-sources at about 4 percent. ***This indicates that mobile sources are likely to account for 85 to 95 percent of the potential cancer risks from outdoor air toxics.***

Figure ES-2: Percent Contributions to Total Cancer Risk at Beacon Hill (year 2000 monitor data)



Uncertainties

The large number of assumptions made in our study reflects the amount of uncertainty and variability associated with the final health risk estimates. It is possible that risk is underestimated because 1) not all air toxics are considered in this analysis, and 2) many chemicals have been shown to accumulate in indoor micro-environments, which could

increase exposure. In addition, potential cancer estimates will underestimate risk for those individuals living near large point sources or "hot spots." Alternatively, risk may be overestimated by assuming that the concentration at the monitor accurately reflects lifetime exposure to ambient pollutants.

It is important to note that this analysis does not evaluate indoor sources of air pollution (i.e., from paints, home furnishings, cleaning products, building materials and other indoor sources). Uncertainties in the toxicity information could also serve to over or underestimate potential risk estimates. These are only a few of the uncertainties associated with this study. A more detailed discussion can be found in Chapter 5 of the full report.

In summary, the information presented in this report uses screening risk estimates as a tool to focus Clean Air Agency attention on those compounds and mixtures that are likely to present the greatest risk of cancer and some non-cancer effects. Consistency among concentrations measured for a limited number of compounds and modeled ambient concentrations, as well as those modeled concentrations that incorporate more exposure data, gives credibility to this ranking effort.

Woodsmoke and diesel soot rank high in potential contributions to cancer and non-cancer risk, higher than other air toxics measured in this study. In addition, volatile organics associated with mobile sources, such as benzene and formaldehyde, contribute significantly to the potential cancer risks from air toxics. Diesel soot, benzene, 1,3 butadiene, and formaldehyde are classified as class A or B carcinogens under the USEPA cancer rating system. This indicates that USEPA is relatively confident that these chemicals probably cause cancer in humans. These chemicals should have high priority during development of an air toxics reduction program for the Puget Sound area. Finally, acrolein appears to present a potential non-cancer risk as well. As stated earlier, the non-cancer health effects associated with the particulate matter related combustion mixtures (e.g., woodsmoke and diesel soot) are not evaluated here, but obviously present serious non-cancer health risks.

Chapter 1: Introduction

Purpose

The purpose of this study is to characterize air emissions and to identify those air toxics and sources that may pose the greatest risks to residents of the Puget Sound area. This analysis uses results from a monitoring study conducted in the greater Seattle/King County area and modeling studies conducted in the four counties in the Puget Sound Clean Air Agency jurisdiction (King, Kitsap, Pierce and Snohomish counties). The United States Environmental Protection Agency (USEPA) performed the modeling in its National-scale Air Toxics Assessment (NATA) project to estimate potential cancer and non-cancer risks associated with the ambient air concentrations of those toxics. In addition, results from a human exposure study provide a general view of the potential exposures and health risks when average or typical human behaviors are considered.

The Puget Sound Clean Air Agency will use the results from this study to evaluate existing air toxics regulations, to focus on compounds of greatest concern, and to identify areas of potential improvements in its air toxics program. These results are intended to provide some general direction to planners and managers who are interested in improving and evaluating existing toxics programs and regulations.

The estimates of cancer and non-cancer health effects should not be viewed as actual cancer or non-cancer cases resulting from air pollution but as an estimate of relative impact of the toxic air pollutants evaluated in order to prioritize agency efforts at reducing exposures. The estimates are based on a combination of average and reasonably conservative or health-protective assumptions. This is expected to lead to risk estimates that are reasonably high for the chemicals included in the analysis, but not worst case.

Overview of Methods

Regulatory agencies typically employ risk-based approaches to evaluate potential health impacts from exposures to toxic chemicals. This study uses basic risk assessment concepts and models to provide a general overview of the potential air toxics problems that could be due to air toxics. However, we have not performed a comprehensive risk assessment, which would include more detailed discussions of toxicity and exposure parameters used to calculate risk estimates.

For the purposes of conducting the screening analysis, potential cancer risks are calculated using the following equation:

Cancer Risk = Exposure x Toxicity

Where: Exposure concentration = annual average (µg/m3)
Toxicity = unit risk for carcinogens (cancer risk/1 µg/m3)

Similarly, non-cancer risks are estimated by calculating a hazard index, using the following equation:

Hazard index (HI) = Exposure/Toxicity

Where: Exposure concentration = annual average (µg/m3)
Toxicity = reference concentration (µg/m3)

Exposure concentrations used to calculate potential cancer and non-cancer health risks were obtained through three different methods. These methods are discussed generally below, and in more detail in chapters 3 and 4 of this report.

Because resources were not available, a complete risk assessment was not conducted. However, the report includes the primary risk assessment components such as a toxicity or dose-response section, an exposure assessment section, and a risk characterization section. It includes a general discussion of the two major types of exposure models

(ASPEN and HAPEM4, discussed later) used to calculate exposure concentrations for two reasons. More comprehensive descriptions of these models were not included for two reasons. First, adequate resources were not available to the agency, and second, these models are described and discussed extensively in technical support documents which accompany the NATA project. However, general descriptions of the model assumptions are included when appropriate, and the supporting documentation is referenced accordingly throughout this document.

Exposure

In this evaluation, three separate methods are used to provide exposure estimates. These include

- Monitored ambient concentrations
- Modeled ambient concentrations
- Modeled "human exposure concentrations" (where human activities and locations are considered in estimating exposures to air pollutants)

Results from each method of predicting exposure are presented and compared with toxicity values and evaluated for potential or relative risk.

Toxicity

Although several different methods are used to estimate exposures, essentially one method is used to evaluate toxicity associated with airborne toxics. In most cases, the USEPA recommended toxicity factors are used as the basis for quantitative dose-response information. These values are usually obtained from the USEPA Integrated Risk Information System (IRIS) database. However, in some cases IRIS values were not available or the USEPA recommended alternative values in the NATA project. In these instances, the alternative values were usually chosen. The basis for each toxicity factor and rationale for any adjustments are included in Chapter 2.

Conclusions and Recommendations

In the last chapter, the results from the different methods used in the evaluation are compared and reasons for discrepancies and similarities are discussed. In addition, the uncertainties and limitations of the evaluation and the impact on the results are described. Finally, recommendations for regulatory priorities are provided.

Chapter 2: Toxicity Estimates

Although several different methods are used to evaluate potential exposures, the same toxicity values are used for each of the analyses. Rather than describe toxicity in each section, toxicity estimates and the details associated with them are described in this section.

Separating Carcinogenic and Non-carcinogenic Impacts

It is important to recognize that toxicity estimates for carcinogens and non-carcinogens are derived through different processes and reflect fundamentally different concepts in toxicity. The toxicity values for non-cancer effects are based on the idea that a threshold exists for these health effects. USEPA believes that carcinogenic effects may not have thresholds, and that any exposure is associated with some corresponding (although very low) risk of disease. Physiological changes leading to cancer may occur over many years or decades.

Carcinogenic health effects are presented as a probability or risk of developing cancer. This can be viewed in two ways. First, the risk concept can be viewed as an additional cancer risk for each exposed individual. For example, a risk of one in one million could be added to the existing cancer risk of one in three (this excludes consideration of genetic or other susceptibilities) for most individuals. USEPA also interprets risk estimates as potential cancer cases over the population of potentially exposed individuals. For example, a one in a million risk can also be viewed as one additional cancer case for every million people exposed to that concentration.[1,2]

[1] USEPA IRIS Glossary defines the unit risk value as **Unit Risk:** The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/L in water, or 1 µg/m^3 in air. The interpretation of unit risk would be as follows: if unit risk = 1.5 x 10^{-6} µg/L, 1.5 excess tumors are expected to develop per 1,000,000 people if exposed daily for a lifetime to 1 µg of the chemical in 1 liter of drinking water.

[2] USEPA also defines "one in a million risk" in the NATA glossary (http://www.epa.gov/ttn/atw/nata/gloss1.html) as follows: **1 in a Million Cancer Risk:** A risk level of 1 in a million implies a likelihood that up to one person, out of one million equally exposed people would contract cancer if exposed continuously (24 hours per day) to the specific concentration over 70 years (an assumed lifetime). This would be in addition to those cancer cases that would normally occur in an unexposed population of one million people. Note that this assessment looks at **lifetime** cancer risks, which

Even if the cancer risks are very small (e.g., one in ten million), they are presented as a probability.

In contrast to carcinogenic health effect evaluation, non-carcinogenic effects are presented as exceeding (or not exceeding) a particular guideline or standard (in this document, a hazard index). The hazard index is a ratio of a concentration deemed to have no adverse effect from a lifetime exposure to that level, divided by the estimated exposure concentration. This non-carcinogen evaluation does not calculate a probability but instead determines whether a particular exposure is below a threshold above which there will be an adverse effect. Levels below the hazard index are deemed to be of no risk. Because of these differences, carcinogenic effects are evaluated separately from non-carcinogenic effects.

Carcinogenic Effects

Carcinogenic effects are commonly measured using a unit risk factor. The unit risk factor is a measure of the potential cancer risk of exposure to 1 microgram per cubic meter of the chemical over a 70-year period.[3] These values are typically derived from animal laboratory studies, although human data from epidemiological or clinical studies are sometimes used. In addition, the unit risk factor is based on the upper 95th percentile of the potency slope, and is therefore considered to be highly conservative or protective of health. In other words, it is unlikely that the potential cancer-risk values underestimate the true cancer risk associated with the specified exposure concentrations, and very likely overestimate the true risk. While this assumption may be true for the specific chemicals under evaluation, it is important to note that lack of information in a number of areas could lead to underestimating or overestimating potential risks. For example, adequate health information on which to base risk estimates is not available for the majority of

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should not be confused with or compared to **annual** cancer risk estimates. If you would like to compare an annual cancer risk estimate with the results in this assessment, you would need to multiply that annual estimate by a factor of 70 or alternatively divide the lifetime risk by a factor of 70. A 1 in million lifetime risk to the public in 1996 was 250 cancer cases over a 70 year period.

[3] See USEPA IRIS definition in footnote #1 above.

chemicals used in commerce.[4] In addition, synergistic and/or antagonistic effects among the chemicals are not considered in these potency estimates. Finally, USEPA typically notes that the cancer risks associated with carcinogens could be as low as zero. Uncertainties associated with the risk estimates are discussed more fully in the last chapter of this document.

In addition to the quantitative evaluation, USEPA also assigns each carcinogen a confidence rating based on the certainty associated with the supporting toxicological and health data. The values in this rating are A through E, with Group A being associated with the greatest certainty of evidence for causing cancer in humans and Group E having evidence that the chemical does not cause cancer in humans.

Toxicity estimates used in this report are listed in Table 2-1 below. Most of the unit risk factors were obtained from the USEPA IRIS database. However, some unit risk factors have been adjusted by USEPA for the NATA project. These values are considered to be more appropriate (for this analysis) than the IRIS values because they are likely to reflect current opinions within USEPA.[5] If the NATA values change in the future, or if USEPA recommends alternative values, this project will be updated to reflect those changes. Currently, however, when differences exist between the IRIS value and the NATA value, the NATA value is used.

Chemicals that are evaluated using the NATA URF, rather than the IRIS value, are noted in Table 2-1 or discussed in more detail below. In addition, two complex chemical mixtures, woodsmoke and diesel particulate matter, thought to be of concern by the Puget Sound Clean Air Agency were evaluated using unit risk values not developed by USEPA. These values and the supporting documentation for each are also discussed below.

[4] National Research Council. ***Toxicity Testing: Strategies to Determine Needs and Priorities***. Steering Committee on Identification of Toxic and Potentially Toxic Chemicals for Consideration by the National Toxicology Program. National Academy Press. Washington DC. 1984.
[5] USEPA. ***National-Scale Air Toxics Assessment for 1996***. Office of Air Quality Planning and Standards. EPA-453/R-01-003. January 2001.

Table 2-1: Unit Risk Factors and Cancer Ratings

Chemical	Unit Risk Factor (risk /µg/m3)	USEPA Cancer Rating	Reference
Acrylonitrile	6.8E-05	B1	IRIS
Benzene	7.80E-06	A	USEPA IRIS file, downloaded 10/22/01
1,3-Butadiene	1.00E-05	B2	USEPA NATA: EPA NCEA[6]
Carbon Tetrachloride	1.50E-05	B2	USEPA IRIS file, downloaded 10/22/01
Chloroform	2.30E-05	B2	USEPA IRIS file, downloaded 10/22/01
Dichloromethane	4.70E-07	B2	USEPA IRIS file, downloaded 10/22/01
Diesel particulate matter (DPM)	3.0E-04	B2	CALEPA/OEHHA
Ethylene dibromide	2.2E-04	B2	USEPA NATA: IRIS
Ethylene dichloride	2.6E-05	B2	USEPA NATA: IRIS
Ethylene oxide	8.8E-05	B1	CalEPA
Hexachlorobenzene	4.6E-04	B2	USEPA NATA: IRIS
Hydrazine	4.9E-03	B2	USEPA NATA: IRIS
1,1,2,2 tetrachloroethane	5.8E-05	C	IRIS
Tetrachloroethylene	5.6E-06	B2	USEPA NATA: CalEPA
Trichloroethylene	2.00E-06	B2	USEPA NATA: CalEPA
Vinyl chloride	8.8E-06	A	USEPA NATA: EPA NCEA
Acetaldehyde	2.20E-06	B2	USEPA NATA: IRIS
Formaldehyde	1.30E-05	B1	IRIS
Arsenic	4.30E-03	B1	IRIS
Beryllium compounds	2.4E-03	B1	IRIS
Chemical	**Unit Risk**	**USEPA**	

[6] EPA National Center for Environmental Assessment.

	Factor (risk /µg/m3)	Cancer Rating	Reference
Cadmium	1.80E-03	B1	IRIS
Chromium compounds (VI)	4.10E-03	A	USEPA NATA: IRIS
Lead	1.20E-05	B2	USEPA NATA: CalEPA
Nickel	1.20E-04	A	USEPA NATA: IRIS
Woodsmoke	1.0E-05		Lewtas et al., 1987

Chromium

The chromium value used here reflects some adjustment for ambient concentrations that may differ between the toxicity study and the ambient measured concentrations. For example, the monitored concentrations of chromium reflect total chromium, while the toxicity value for chromium is reported to be primarily for chromium VI.[7] However, the chromium VI unit risk value is based on human epidemiological data obtained from chromate production workers exposed to total chromium, but with "no less than one seventh" due to chromium VI (approximately 14% chromium VI of the total chromium measured in the ambient air).[8] Ambient urban air is not expected to have chromium VI contributions as high as those observed in chromate production plants or other industrial facilities.[9] Therefore, some adjustment must be made for the differing percentages of chromium VI between the ambient urban measurements and the ambient levels in the occupational studies.

USEPA recognized this discrepancy in the NATA evaluation, and adjusted the value accordingly. To account for difference between the ratios, the chromium

[7] USEPA IRIS file for Chromium. Downloaded 9/27/01.
[8] USEPA IRIS file for Chromium.
[9] Menne MG, W Koot, EM van Putten, R Ritsema, S Piso, T Knol, F Fortezza, and JG Kliest. Hexavalent chromium in ambient air in the Netherlands. *Results of measurements near wood preservation plants and at a regional site*. Report no 723101031. National Institute of Public Health and the Environment. April 1998.

values are conservatively assumed to contain approximately 34% of the highly toxic chromium VI.[10] This is very likely an overestimate of the amount of chromium VI in the ambient air, and therefore overestimates the potential cancer risk. However, since it is expected to be more accurate than using the unadjusted chromium unit risk factor, and is consistent with NATA, it is used in the following analyses.

In addition to evaluating the potential cancer risks from a number of individual chemicals, we evaluated both woodstove and diesel particulate matter emissions as mixtures.

Woodsmoke

Toxics adsorbed to particles can also present significant risks to human health. The mixture that results from wood combustion contains thousands of compounds, an unknown number of which adsorb to particle surfaces. The particles carry these substances deep into the lung during inhalation. While specific knowledge of the role of these adsorbed particles is not known, it is hypothesized that the presence of such toxics plays a role in particle health effects. For example, polynuclear aromatic hydrocarbons (PAHs) are known to adsorb to combustion particles from all sources. Because relatively little is known about the cumulative toxicity of the multiple toxics present in certain combustion mixtures, we prefer to use unit risk factors for these mixtures as a whole instead of individual unit risk factors for carcinogens which are available for relatively few pollutants in the woodsmoke mixture. The unit risk factor for woodsmoke was developed through a comparative potency method where the mutagenicity and tumor initiating potency from particles emitted from several sources (e.g., diesels, woodsmoke and gasoline-powered automobiles) are systematically evaluated. Mutagenicity tests are conducted on different segments of the total mixtures. Segments containing higher mutagenic potencies are further divided into groups and tested until the components or segments with the highest

[10] USEPA, 2001.

potencies are identified.[11] The unit risk value used in this analysis for woodsmoke is listed in Table 2-1.

Diesel Particulate Matter Unit Risk Factor

The carcinogenicity of diesel particulate matter is widely recognized by a number of health agencies including the USEPA,[12] CalEPA,[13] the US Department of Health and Human Services,[14] and the International Agency for Research on Cancer (IARC).[15] Because USEPA has not yet developed a unit risk factor for diesel particulate matter, the CalEPA value is used in this analysis.[16] This value is also listed in Table 2-1.

Although it has not identified a final unit risk factor for diesel particulate matter, USEPA states firmly that diesel particulate matter is a B1 or probable human carcinogen. In absence of a confirmed URF, USEPA provides a range of potential cancer risks associated with environmental exposures (i.e. exposure levels typically experience by the general population) in a section 8.4, entitled "Perspectives on Cancer Risk" of their Health Assessment Document. USEPA estimates this range to be approximately 4E-05 to 2E-03. This range assumes average environmental exposures of 0.8 - 4.0 $\mu g/m^3$ over a lifetime.[17] These ranges are comparable to the unit risk estimate calculated by CalEPA. For example, assuming an environmental exposure of 1 µg/m3, the range recommended by USEPA could be approximately 5E-05 to 2E-03.[18] The unit risk

[11] Lewtas J. *Genotoxicity of Complex Mixtures: Strategies for the Identification and Comparative Assessment of Airborne Mutagens and Carcinogens from Combustion Sources*. Funda and Appl Tox 10, 571-589. 1988.
[12] USEPA. ***Health Assessment Document for Diesel Exhaust***. Office of Research and Development. EPA/600/8-90/057E. July 2000.
[13] CalEPA/OEHHA. *For the "Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant. Part B: Health Risk Assessment for Diesel Exhaust*. May 1998.
[14] National Toxicology Program. Public Health Service, US Department of Health and Human Services**.** ***9th Report on Carcinogens*.** Revised January 2001.
[15] International Agency for Research on Cancer (IARC***). IARC Monograph on the Evaluation of Carcinogenic Risks to Humans. Vol. 46: Diesel and Gasoline Engine Exhausts***. 1989.
[16] CalEPA, 1998.
[17] USEPA 2000.
[18] Assuming an environmental exposure of 1 µg/m3, an EO ratio of 1 to 365 can be calculated using the "broad concentration range" for occupational exposures listed in Table 8-1 of the USEPA 2000 report.

factor recommended by CalEPA, 3E-04, is around the mid-point of USEPA's range. This suggests that the CalEPA unit risk factor is not as highly conservative as USEPA's high-end estimates.

Non-carcinogenic Effects

Many chemicals also have non-cancer health effects associated with them. Non-carcinogenic effects are presumed to have a threshold of exposure below which no effect occurs, although this is not always the case (e.g., fine particulate matter). Non-carcinogenic effects from air exposures are evaluated using reference concentrations. Reference concentrations (RfCs), like unit risk factors, are based on animal or human studies. RfCs are derived by examining the literature to find a critical study, which is defined as a well-designed chronic exposure study that has identified the non-carcinogenic adverse effect that occurs at the lowest level of inhalation exposure. The no-observable-effect-level (NOEL) or a lowest-observable-adverse-effect level (LOAEL) from animal or human studies is determined. Adjustments for exposure times are made to extrapolate exposures to 24 hours, seven days per week, and conversion to units of mg/m3 are made. A human equivalent concentration is calculated by considering the nature of the contaminant and its behavior in inhaled air; the region of the respiratory system impacted; and the surface area and respiratory rate of the test organism, relative to the same parameters in humans. This concentration is then divided by factors of 10 to account for uncertainties such as extrapolating from animals to humans, from healthy adult individuals to sensitive individuals, or from sub-chronic to chronic exposures. The RfC also include confidence statements that speak to the extent and quality of the database, and the certainty of the RfC, based on supporting literature aside from the critical study.

As a result of these types of derivations, the RfC is also considered to be highly conservative or protective of human health. Similar to the unit risk factors used for carcinogens, USEPA considers the RfC to be unlikely to underestimate potential risks to

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Multiplying these values by the 2% excess risk due to diesel particulate matter exposures results in 5E-05 to 2E-03, also assuming an EO ratio of 10, rather than 1 (1 seems unlikely).

humans. It is important to recognize that many chemicals can have a variety of effects that occur at different levels of exposure. The RfC only looks at the effect that occurs at the lowest level of exposure. The assumption is made that protection at this level also provide protection at the higher doses as well.

To determine a hazard index for these chemicals, the RfC is compared to the annual average or median concentration for each of the three exposure data sets (e.g., the monitoring results, the ambient modeling results, and the human exposure modeling results). Although it may be useful to compare the RfC to an upper-bound concentration for a given area, this value may overestimate exposures over the lifetime of the exposed individual. RfCs used in this evaluation are listed in Table 2-2 below. The name of the chemical is listed with the RfC value, the uncertainty factors and modifying factors used in calculating the RfC, the critical effect, and the source for the information. The information used in the analysis and listed in the table was taken from the USEPA NATA report.

The non-cancer health effects associated with diesel particulate matter and woodsmoke, and the fine particulate fraction of these mixtures specifically are not included in this evaluation. Non-cancer health effects associated with fine particles, such as morbidity related effects such as increased asthma attacks, upper respiratory irritation, and increased mortality are analyzed elsewhere.

Table 2-2: ***Reference Concentrations for Air Toxics***

Chemical	RfC (mg/m3)	UF x MF	Target Organ for Critical Effect	Source
Acetaldehyde	9.0E-03	1000	Nasal epithelium	IRIS
Acrolein	2.0E-05	1000	Nasal epithelium	IRIS
Acryolonitrile	2.0E-03	100	Nasal epithelium	IRIS
Arsenic and compounds	3.0E-05	1000	Teratogenic effects	Cal EPA
Benzene	6.0E-02	10	Blood, bone marrow	Cal EPA
Beryllium Compounds	2.0E-05	10	Lung	IRIS
1,3 butadiene	8.0E-03	300	Reproductive system	Cal EPA
Cadmium compounds	2.0E-05	30	Kidney	Cal EPA
Carbon Tetrachloride	4.0E-02	300	Liver	Cal EPA
Chloroform	9.8E-02	100	Liver, kidney	ATSDR
Chromium compounds	1.0E-04	90	Respiratory tract	IRIS
1,3 Dichloropropene	2.0E-02	30	Nasal epithelium	IRIS
Ethylene dibromide	8.0E-04	100	Reproductive system	Cal EPA
Ethylene dichloride	2.4E-00	90	Kidney	ATSDR
Ethylene oxide	3.0E-02	100	Blood	Cal EPA
Formaldehyde	9.8E-03	30	Respiratory tract	ATSDR
Hexachlorobenzene	3.0E-03	100	Teratogenic effects	Cal EPA
Hydrazine	2.0E-04	300	Liver, thyroid	Cal EPA
Lead compounds	1.5E-03	1	Central nervous sys.	NAAQS
Manganese compounds	5.0E-05	1000	Central nervous sys.	IRIS
Mercury compounds	3.0E-04	30	Central nervous sys.	IRIS
Methylene chloride	1.0E+00	30	Liver	ATSDR
Nickel compounds	2.0E-04	30	Respiratory tract	ATSDR
Propylene dichloride	4.0E-03	300	Nasal epithelium	IRIS
Tetrachloroethylene (perc)	2.7E-01	100	Central nervous sys.	ATSDR
Trichloroethylene	6.0E-01	100	Central nervous sys.	Cal EPA
Vinyl chloride	1.0E-01	300	Liver	Cal EPA

Chapter 3: Air Toxics Monitoring Information

Ambient air concentrations have been collected through several different studies. The primary study is the *Seattle Air Toxics Monitoring Study*, conducted during the 2000 and 2001 calendar years. Complete data are available for the 2000 calendar year, but not for the 2001 calendar year. In addition to this study, two other studies provided ambient air concentrations of woodsmoke and diesel particulate matter in the Seattle area. These studies include *Annual Average Source Contributions to PM2.5 at Three Residential Sites in the Puget Sound Region* and a recent study conducted by Maykut, Larson and Lewtas, referred to here as the *Positive Matrix Factorization project*. These studies are described and discussed in more detail below.

Seattle Air Toxics Monitoring Study

The *Seattle Air Toxics Monitoring Study* was conducted as a collaborative effort among three agencies (USEPA, the Washington State Department of Ecology, and the Puget Sound Clean Air Agency) during 2000 and 2001. The purposes of this study were to provide information on the spatial and temporal variability of ambient air toxics, to evaluate modeling results obtained from the NATA project, and compare results to other urban areas in the United States. The objective of this study was to quantify the urban air toxics such as VOCs, carbonyl, and metal species on a regular basis at several surface sites in Seattle.

Seattle was originally selected by USEPA for this monitoring study as one of four cities nationwide. USEPA initiated this air toxics monitoring project as part of its overall National-scale Air Toxics Program (NATA).[19] The federal Clean Air Act mandates USEPA to determine a subset of the 188 urban hazardous air pollutants (HAPs) that potentially pose the greatest risks in urban areas. USEPA identified a total of 33 urban

[19] USEPA. Peer Review Draft for the Science Advisory Committee: *Air Toxics Monitoring Concept Paper*. Office of Air Quality Planning and Standards. February 2000.

HAPs in their 1995 ranking analysis,[20] and developed concurrent monitoring and modeling programs (e.g., NATA) to evaluate potential exposures to these top-ranked 33 HAPs.[21] These 33 are discussed more fully in Chapter 4.

Of the 33 HAPs identified by USEPA, a total of 18 HAPs were monitored at two locations in the Seattle area during the 2000 calendar year, and at six sites during the 2001 calendar year. The remaining 15 HAPs were not monitored because they were considered less stable or lacked approved collection and/or analytical techniques. Twenty-four-hour integrated air samples were collected every six days at each site.[22] Such collection techniques provide that every day of the week is sampled over time.

Site Locations and Selected Pollutants

A total of six sites were selected to represent the Seattle urban area. Two sites were monitored during the 2000 calendar year, and four additional sites (for a total of six sites) were monitored during the 2001 calendar year (see Figure 3-1).

The two sites monitored during 2000 were Beacon Hill and Georgetown. The first site represents a typical urban residential area. Beacon Hill (Fig. 3-1: 3) was selected to represent this type of area because it has a relatively high population density and is impacted by a mix of urban source categories. For example, it is located near the Interstate 90 and Interstate 5 interchange, and is also impacted by local sources. However, it is more significantly impacted by urban residential sources such as mobile exhaust and woodsmoke. The second area was selected to represent potentially maximum concentrations near an industrial area. This site is located in the Georgetown neighborhood (Fig. 3-1: 4). It is impacted by several large industrial sources, as well as an airport. It is also impacted by mobile sources from Highway 99 and nearby roadways. This neighborhood is located in the Duwamish industrial valley.

[20] USEPA. *Ranking and Selection of Hazardous Air Pollutants for Listing Under Section 112j of the Clean Air Act Amendments of 1990, Technical Support Document*, July 28, 1999.
[21] USEPA. *National Air Toxics Program: The Integrated Urban Strategy Report to Congress*. Office of Air Quality Planning and Standards. EPA-453/R-99-007. July 2000.

Four more sites were added for the 2001 calendar year. These sites include: Lake Sammamish (Fig. 3-1:6) for an urban background site, Maple Leaf (Fig.3-1: 2) for a typical urban residential site, Sea Tac (Fig. 3-1: 5) for a site that is highly impacted by mobile sources, and Lake Forest Park (Fig.3-1: 1) for an area impacted by woodstoves and mobile sources.

It is also important to note that two sites are located near airports. The SeaTac monitor is located north of the Seattle-Tacoma International Airport, a major airport which serves the Puget Sound area. The Georgetown site is also located near an airport which serves a number of commercial industries including The Boeing Company, a major aerospace company. The potential impact of these airport emissions on the monitored concentrations are discussed in the latter sections of this chapter. The location of each of the six sites is shown on the map below.

[22] Washington Department of Ecology. *Urban Air Toxic Measurements in Seattle*. Conducted by the Laboratory for Atmospheric Research, Washington State University, Pullman WA. Contract # C0000060. Project Officer: John Williamson. Bellevue, WA May 2001.

Figure 3-1: Air Toxics Monitor Locations

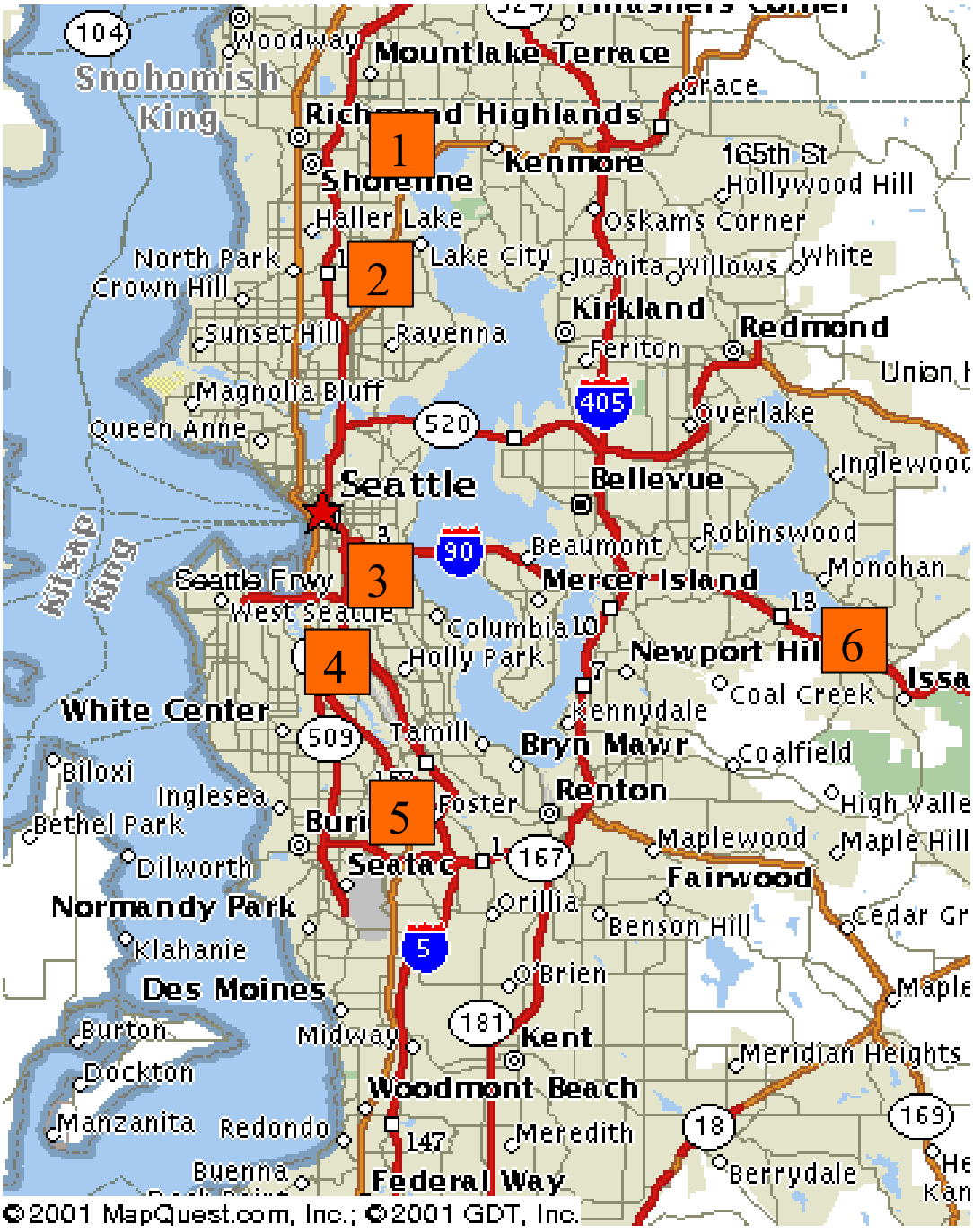


Table 3-1: Monitored Urban Air Toxic Pollutants (18 total)

CAS No.	*VOCs*	*CAS No.*	*Metals*
71432	Benzene	7440382	Arsenic
7440439	1,3 butadiene	Total compounds	Beryllium
56235	Carbon Tetrachloride	Total compounds	Cadmium
67663	Chloroform	Total compounds	Chromium
75092	Dichloromethane	7439921	Lead
78875	1,2 dichloropropane	Total compounds	Manganese
127184	Tetrachloroethene	7440020	Nickel
79016	Trichloroethene		*Carbonyls*
75014	Vinyl chloride	75070	Acetaldehyde
		50000	Formaldehyde

Woodsmoke Concentration Estimates

In addition to risks from ambient air toxics, ambient concentrations of woodsmoke have long been recognized as potentially carcinogenic and contribute substantially to ambient particulate-matter concentrations in the Puget Sound area.[23,24] In an effort to quantify potential risks from woodsmoke, ambient woodsmoke concentrations are multiplied by a woodsmoke unit risk factor (see methods in Chapters 1 and 2). Two studies have been conducted that estimate ambient woodsmoke concentrations in the Puget Sound region: *Annual Average Source Contributions to PM2.5 at Three Residential Sites in the Puget Sound Region* and a recent study conducted by Maykut, Larson and Lewtas, referred to here as the *Positive Matrix Factorization Project*.

[23] Lewtas J. *Genotoxicity of Complex Mixtures: Strategies for the Identification and Comparative Assessment of Airborne Mutagens and Carcinogens from Combustion Sources.* Fundamental and Applied Toxicology 10, 571-589. 1988.

[24] Yuen Po-Fat and Tim Larsen. *Annual Average Source Contributions to PM2.5 at Three Residential Sites in the Puget Sound Region.* Environmental Engineering and Sciences, Department of Civil Engineering University of Washington, Seattle WA. Received September 15, 1993 at the Puget Sound Clean Air Agency.

Annual Average Source Contributions to PM2.5

In the first study, the Puget Sound Clean Air Agency contracted with the University of Washington's Engineering Department to conduct an ambient monitoring study of woodsmoke in three residential areas in the Puget Sound Region.[25] This study was conducted between January 1991 and January 1992, and the final report received by the Puget Sound Clean Air Agency (formerly Puget Sound Air Pollution Control Agency) in September 1993.

Ambient monitoring was conducted at three residential locations: Marysville, Lake Forest Park, and Puyallup. Sampling at the Lake Forest Park location was done at the Puget Sound Clean Air Agency site, and is close to the location of the current 2000 and 2001 Lake Forest Park monitor. The Marysville monitor was located at the main fire station approximately one mile north of the Marysville City Hall. The Puyallup monitoring site was located at the Fire District No. 9 Fire Station in South Puyallup. PM2.5 monitoring was accomplished using Harvard impactors.

Sampling was conducted each day using 15-minute intervals, and a total of 53 weekly samples were collected over the year. Each weekly sample represented a composite of 84 15-minute samples. These samples were combined for annual average PM2.5 concentration at each of the three sites.

Contributions from woodsmoke, mobile sources, and atmospheric sulfate were derived from the total PM2.5 concentrations using three different models. The authors conclude that the model referred to as the effective variance weighted chemical mass balance (EV-CMB) provides the best estimate of annual average woodsmoke concentrations, and that woodsmoke particles are estimated to contribute approximately 70 to 75% of the total PM2.5 concentrations in these areas.[26] Woodsmoke concentration estimates are presented in Table 3-2.

[25] Yuen and Larsen. 1993.
[26] Yuen and Larson., 1993.

Table 3-2: Estimated Woodsmoke Concentrations (µg/m3) at 3 Sampling Sites

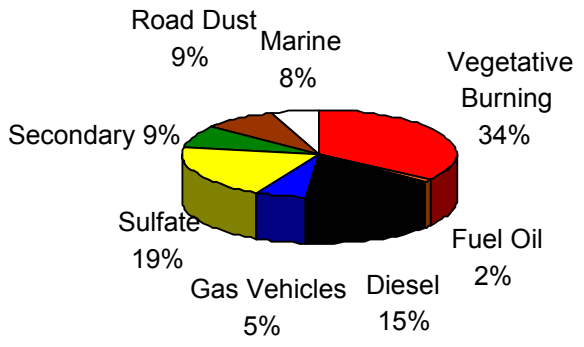
Location	*Annual Mean Woodsmoke Concentration (µg/m3)*
Lake Forest Park	11
Marysville	12
Puyallup	11

It is possible that current ambient concentrations contributed by woodsmoke are somewhat less than those reported in the 1993 study. However no data are currently available that would allow any adjustments to the estimates discussed above.

Positive Matrix Factorization (PMF) Modeling

Speciated data from Seattle's Beacon Hill PM2.5 monitoring site were analyzed by the Positive Matrix Factorization (PMF) source apportionment model to identify the major sources of PM2.5 and organic carbon in Seattle's air. The sources identified by the PMF model are (in descending order of importance): vegetative burning such as wood-burning fireplaces and yard waste (indoor and outdoor), motor vehicles (gasoline and diesel), secondary sulfate, secondary nitrate, soil, and marine sea salt (see Figure 3-2).

Figure 3-2: PMF SOURCE APPORTIONMENT - SEATTLE BEACON HILL 1996-99 USING CARBON FRACTIONS



The average concentration of PM2.5 at Beacon Hill from March 1996 through February 1999 was 9 $\mu g/m^3$. This translates to average annual concentrations of approximately 3 $\mu g/m^3$ for woodsmoke and 1.4 $\mu g/m^3$ of diesel particulate.

Diesel Particulate Matter Concentrations

Diesel particulate matter is also widely recognized as a potent carcinogen. Although ambient concentrations were not available from each of the six sites in the monitoring study, diesel particulate matter was estimated from the PM2.5 monitor located at the Beacon Hill site for three years using the Positive Matrix Factorization (PMF model).[27] The ambient annual concentration of diesel particulate matter at the Beacon Hill site is estimated to be 1.4 µg/m3. This value compares well with the modeled estimate for King County of 1.77 µg/m3.[28]

Estimated Potential Cancer Risks from Six Monitoring Sites

Potential cancer risk estimates for each chemical at each of the six sites of the Seattle Air Toxics Monitoring Study are presented in Figure 3-3 and Table 3-3. In addition, cancer risks for the average concentrations across all 6 sites are also presented. These values are presented as individual cancer risk per million (over a 70-year lifetime) or potential cancer cases per million people exposed over a 70-year exposure period.

It is important to recognize that these cancer risk estimates are based on the assumption that individuals (either one or many in an exposed population) are exposed to this average concentration for their entire lifetime or an exposure period of 70 years. These concentrations may or may not represent actual annual average exposures for individuals throughout the Seattle population. For example, it is highly unlikely that an individual would spend an entire 70-year period outside near a particular monitor. However, it is possible that the ambient average concentration is a reasonable approximation of the annual average human exposure since both individuals and contaminants move through

[27] Maykut N, J Lewtas and T Larson, presented at Regional Haze and Global Radiation Balance Conference. Bend, Oregon. October 2001.
[28] USEPA *National Air Toxics Assessment Final*. Downloaded ASPEN results for Washington State, January 2002.

various micro-environments. These issues will be discussed more fully later in sections pertaining to human exposure modeling results and in the conclusions and discussion section.

Figure 3-3: Potential Cancer Risks Associated with Seattle Monitoring Study Results

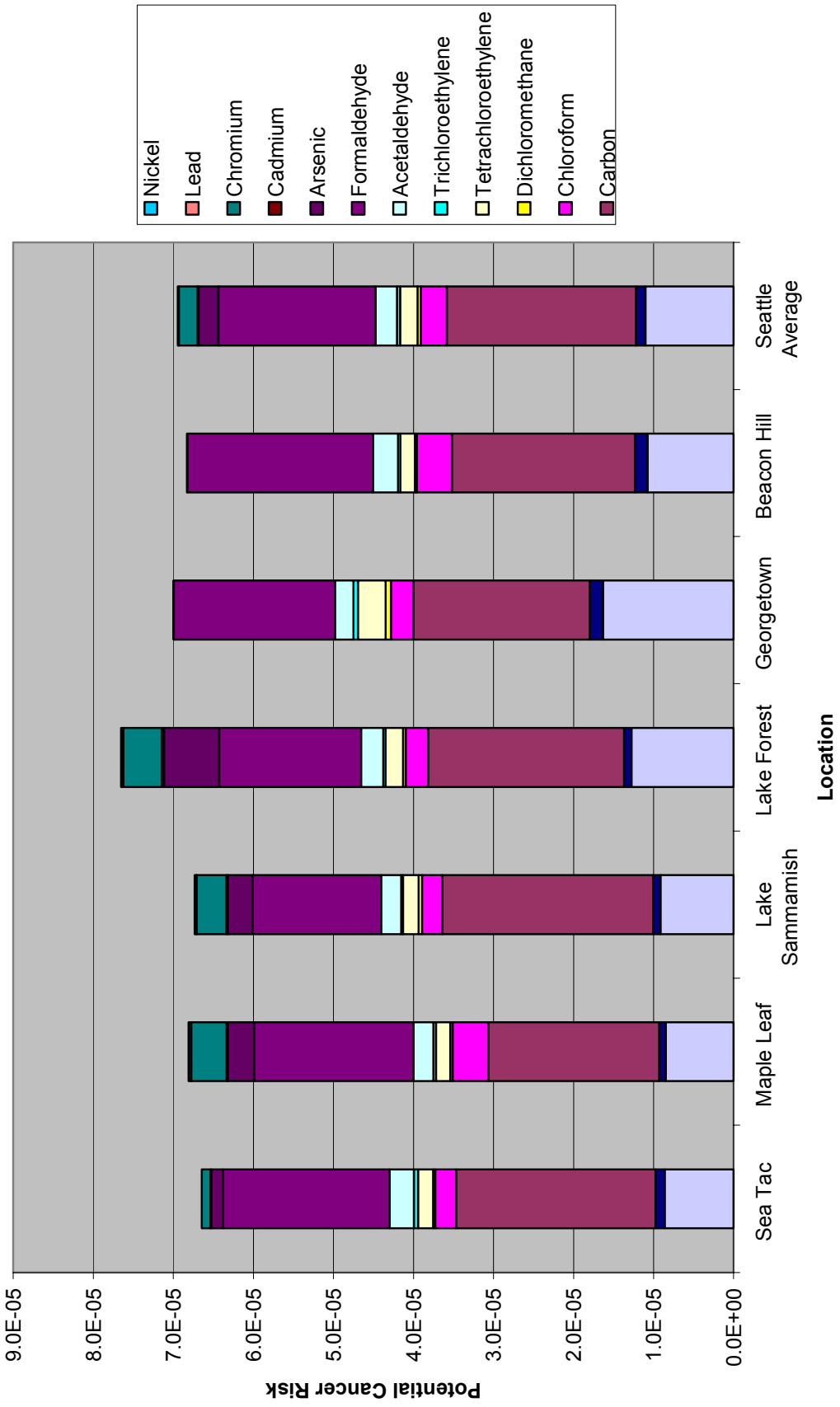


Table 3-3: Estimated Cancer Risks Per Million Associated with Seattle Air Toxics Monitoring Study Results

	Sea Tac Cancer Risk	Maple Leaf Risk	Lake Sammamish Risk	Lake Forest Risk	Georgetown Risk	Beacon Hill Risk	Seattle Average Risk
Benzene	8.6	8.5	9.1	13	16	11	11
1,3-Butadiene	1.1	0.8	0.95	0.93	1.6	1.6	1.2
Carbon Tetrachloride	25	21	26	24	22	23	24
Chloroform	2.6	4.5	2.5	2.9	2.8	4.4	3.3
Dichloromethane	0.26	0.32	0.43	0.31	0.64	0.24	0.37
Tetrachloroethylene	1.9	1.8	1.9	2.2	3.5	1.8	2.2
Trichloroethylene	0.52	0.32	0.27	0.30	0.57	0.30	0.38
Acetaldehyde	3.0	2.5	2.5	2.8	2.3	3.1	2.7
Formaldehyde	21	20	16	18	20	23	20
Arsenic	1.5	3.3	3.1	6.9	6.3	3.9	2.5
Cadmium	0.090	0.17	0.20	0.29	0.001	0.00076	0.13
Chromium	1.1	4.4	3.7	4.8	0.015	0.0063	2.3
Lead	0.000028	0.0058	0.0064	0.058	0.00013	0.000040	0.03
Nickel	0.00056	0.25	0.2	0.25	0.00037	0.00023	0.12
Total Estimated Cancer Risk	**66**	**68**	**67**	**77**	**70**	**68**	**69**

As indicated on the table and figure, the cancer risk estimates are quite similar among the six Seattle sites, and no individual site appears to have consistently higher risks than the others. Total estimated cancer risks for all chemicals monitored in the study range from 6.6 to 7.7 in one hundred thousand. Cancer risk estimates for the individual chemicals range from very low to 2.6 per one hundred thousand for carbon tetrachloride at Lake Sammamish. No analyses were available to evaluate whether the differences in measured concentrations at various sites were statistically significant.

It appears that Lake Forest Park has the highest monitored concentrations, and therefore a slightly higher risk than the other five monitor locations. This appears to be due to a brief spike in arsenic concentrations monitored at the site. It is not clear why this spike occurred, although a variety of reasons are possible. Some possible explanations include brief periods of wind-blown dust or contaminated wood burning in the area.

Although carbon tetrachloride is a significant contributor to the cancer risk estimates, it is important to note that this chemical has been banned in the Puget Sound area for some time. These monitored concentrations may reflect emissions that are not currently reported or previous contamination that is extremely persistent.

Total excess cancer risk estimates for *mobile source* related chemicals (lead, benzene, 1,3 butadiene, formaldehyde, and acetaldehyde) range between 30 and 40 per million (see Figure 3-4). This estimate does not include all air toxics that are emitted from mobile sources, only the ones that were measured in the monitoring study. Assuming the remaining chemicals are primarily from *stationary sources*, the cancer risk estimates for these sources range from 5 to 20 per million as well (see Figure 3-5). Carbon tetrachloride is not included in either of these graphs because its source has not been clearly identified, although it contributes significantly to the total cancer risk. The total cancer risk for all chemicals monitored in the 2000 and 2001 studies ranges from approximately 66 to 77 per million over a 70-year exposure period for these chemicals. The total risk for the Seattle average is approximately 70 per million over a 70-year exposure period. These risk estimates are based on the assumption that concentrations

observed in this monitoring study will be constant for the assumed 70-year exposure period and that exposures to ambient air reflect the types of exposures that are occurring over the duration.

It is reasonable to expect that the emissions from the two airports could impact the SeaTac and Georgetown monitors. However, the results do not reflect significantly higher pollutant levels at these locations when compared with other monitors. It is possible that the airport emissions do not significantly impact the monitor locations because they do not extend far beyond the airport area. It is also possible that the pollutants of concern at the airport are not those included in the monitor study.

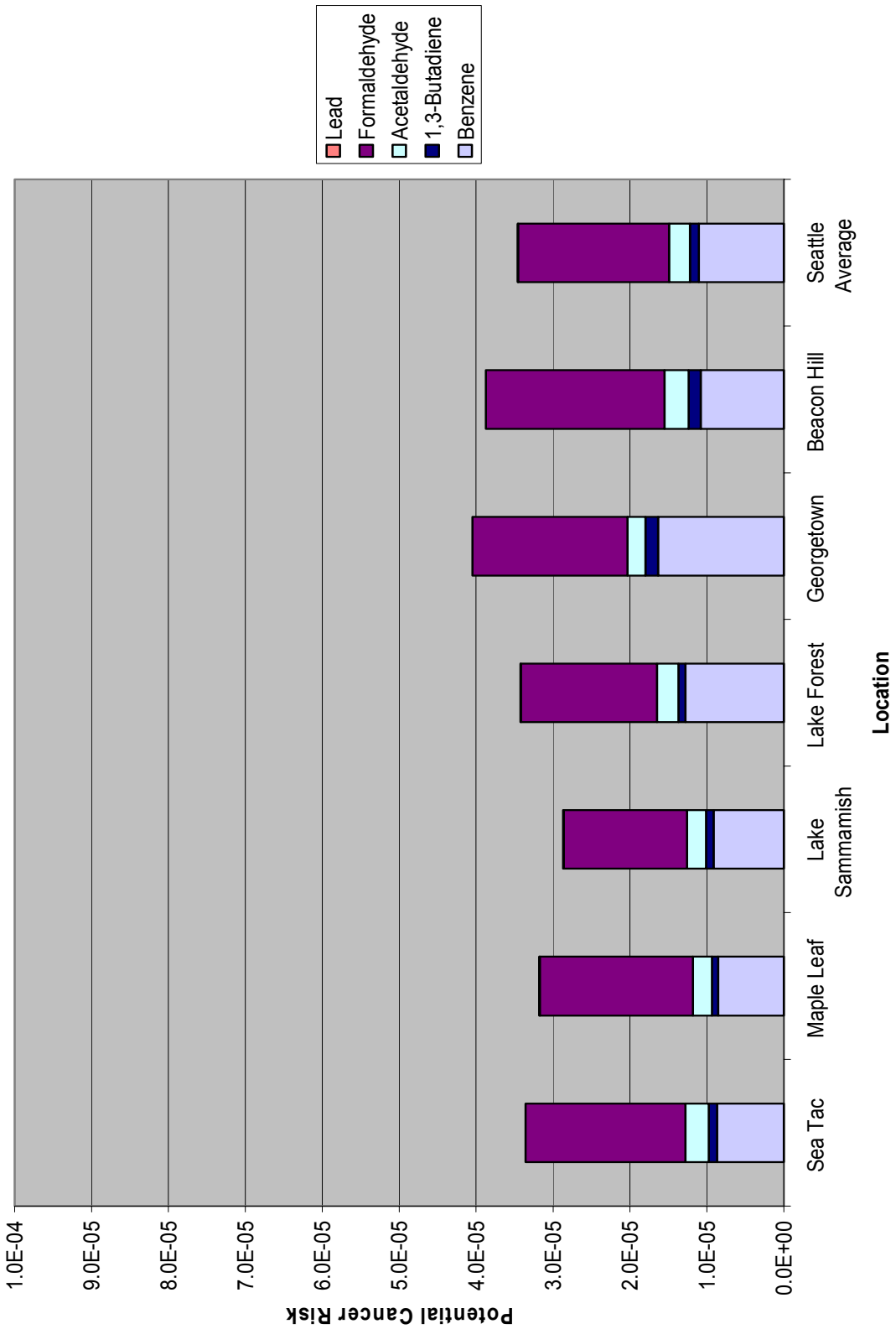


Figure 3-4: Mobile Source Related Chemicals

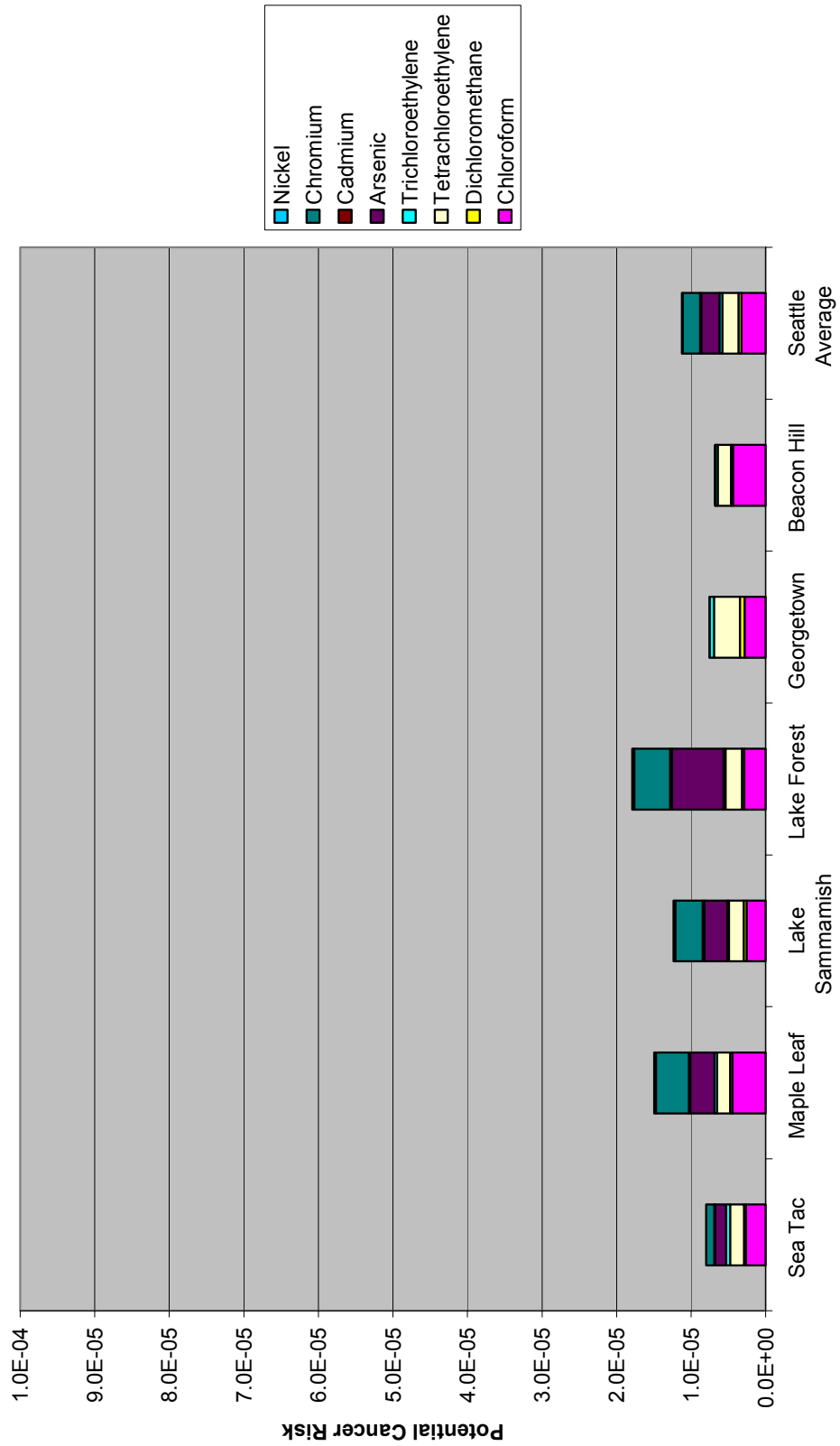


Figure 3-5: Stationary Source Related Chemicals

As described above, cancer risk estimates for these six sites are based on approximately six months of monitored data. However, a full year of monitored data is available for the 2000 calendar year for the Beacon Hill and Georgetown sites. Cancer risk estimates for the full year of data at these two sites are calculated and presented in the charts below (see Figure 3-6).

Potential cancer estimates are similar to those predicted by the six months of monitoring data from the 2001 data, but overall appear higher. For these 15 carcinogens combined, the risk estimates range from 90 to 120 per million over 70 years.

In addition, the same chemicals (benzene, carbon tetrachloride, and formaldehyde) are responsible for most of the overall cancer risks in both locations (see Figure 3-7).

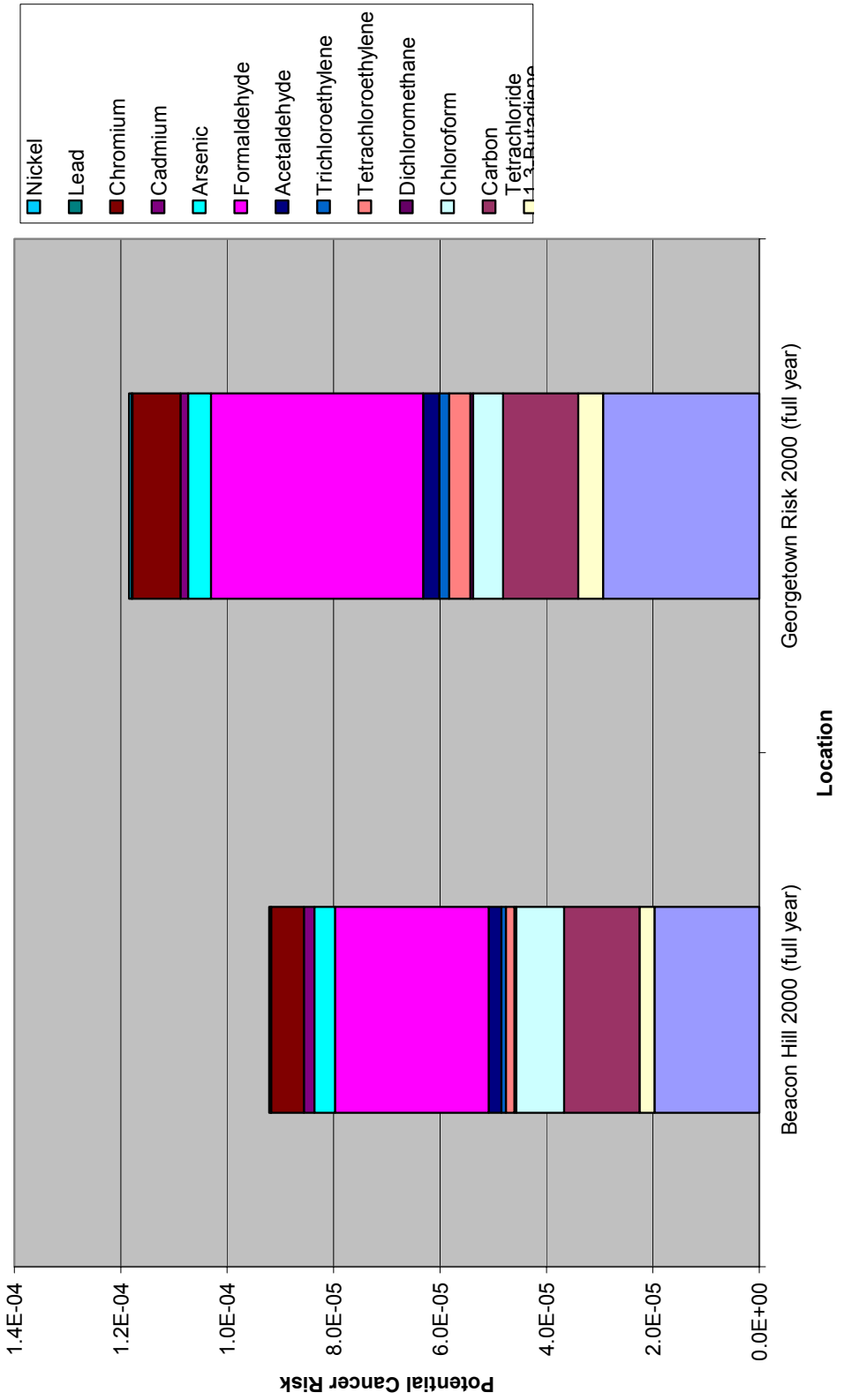


Figure 3-6: Comparing Cancer Risks at 2 Sites for the 2000 Calendar Year

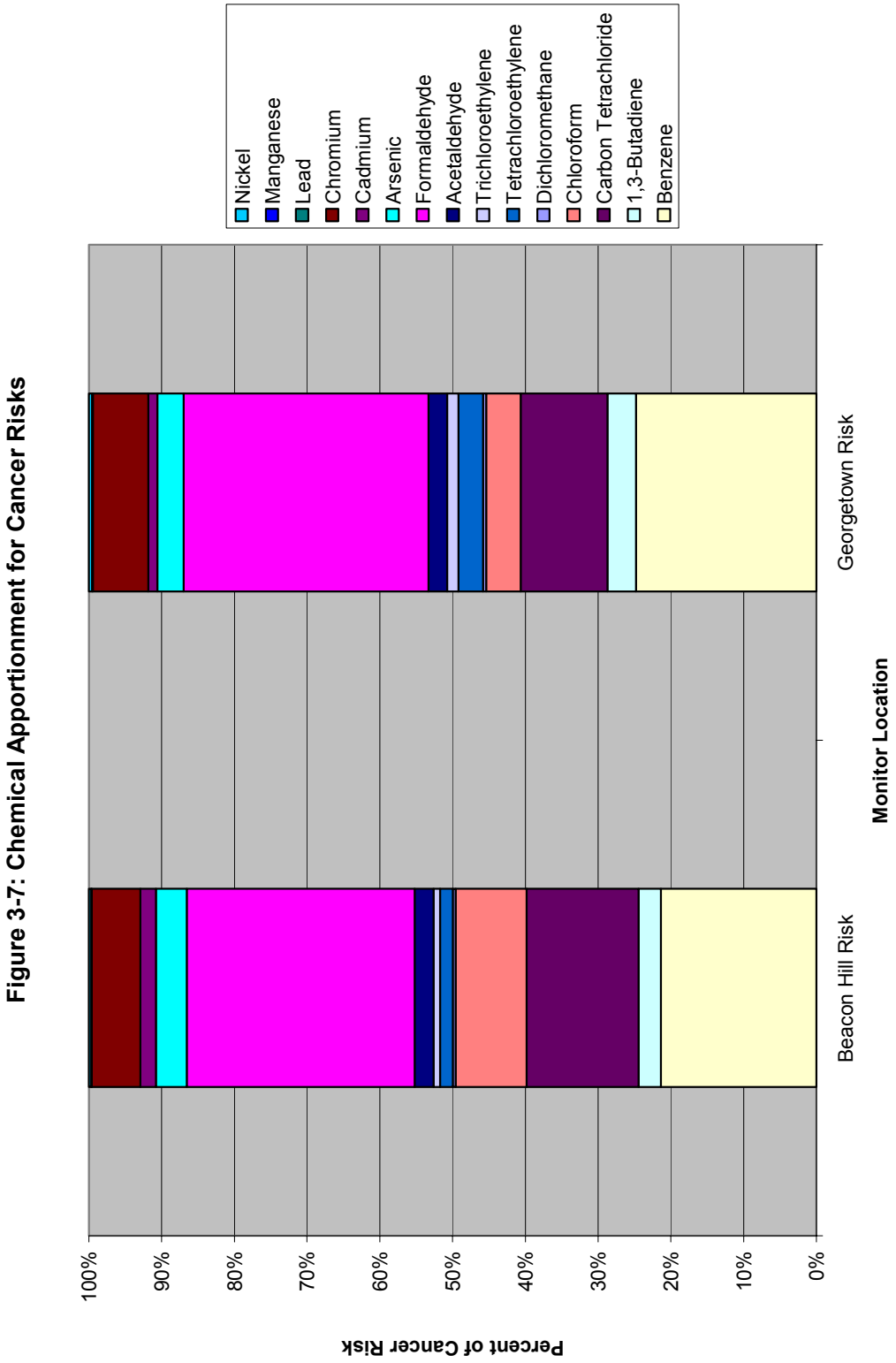


Figure 3-7: Chemical Apportionment for Cancer Risks

The chemicals that pose the greatest risks primarily include those chemicals associated with mobile sources. Similar to Beacon Hill, the Georgetown risks are dominated by the mobile source chemicals benzene, formaldehyde, and 1,3 butadiene. However, the individual risk estimates from these chemicals are slightly higher than those estimated at the Beacon Hill site. This may reflect the fact that the Georgetown monitor is located in the Duwamish Valley where contaminants may readily accumulate during winter inversion conditions.

It is important to note that these monitors are placed in areas that are not expected to be heavily impacted by a large industrial source or "hotspot," except for possibly the Georgetown site. The annual average from the selected monitor locations are expected to reflect general urban settings such as an urban residential area, or an urban industrial area. A few chemicals that are associated with industrial point sources, such as chromium and trichloroethylene, are higher in the Georgetown area. These chemicals probably reflect more general industrial uses of paints, solvents and chrome plating rather than one specific industrial source.

It is also possible that the risk estimates at the six sites will be different when the full year of data is analyzed. For example, when the data sets are compared at each site, the risks appear greater with the full year of data than for spring and summer alone. This may reflect year to year variation or may reflect seasonal variation. The 2001 data were collected between late February and July of 2001, and do not include a significant portion of the winter months. A more complete comparison will be made when the full monitoring study becomes available later this year.

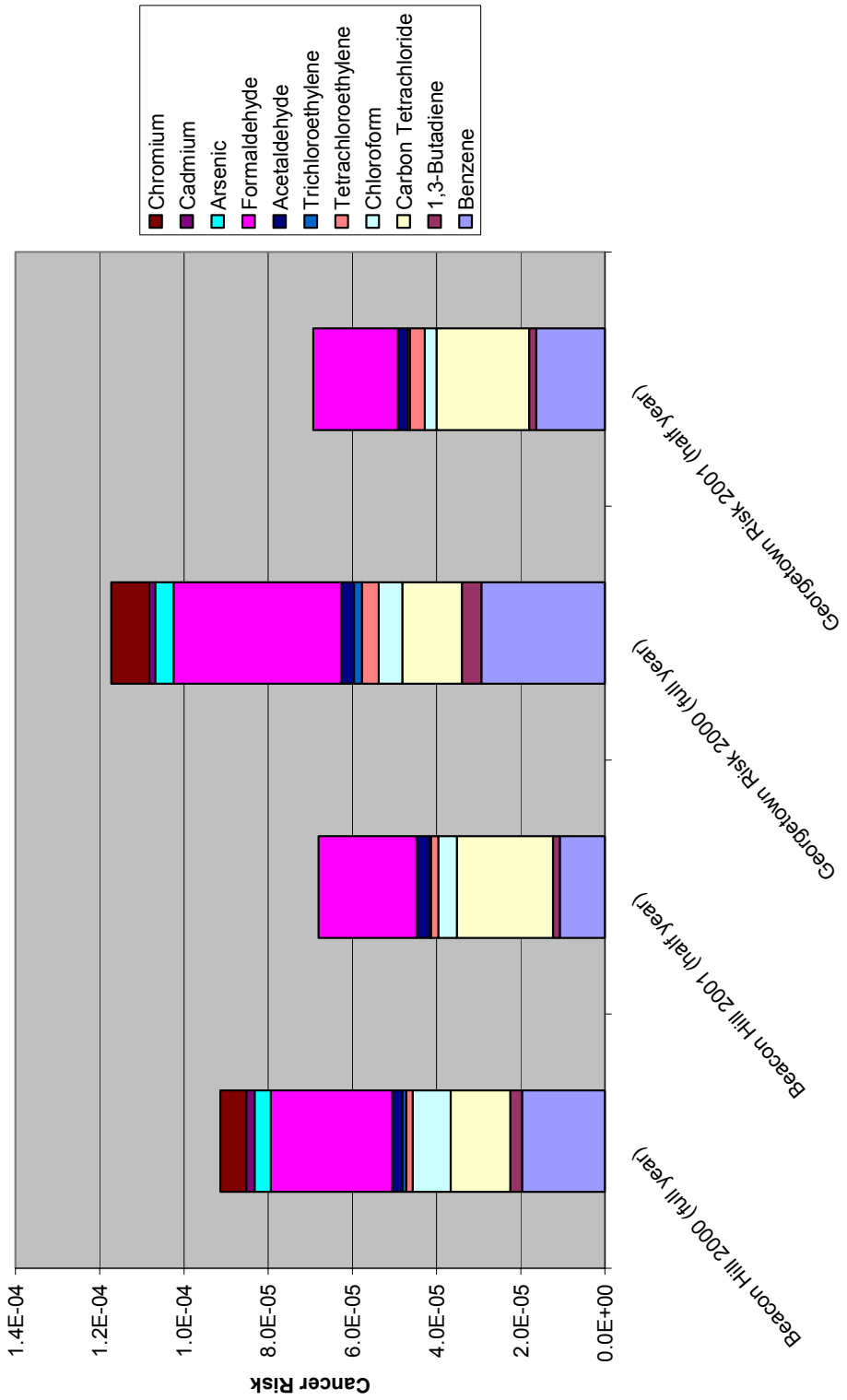


Figure 3-8: Comparing Risks Between 2000 with 2001

In addition to the air toxics measured in the 2000 and 2001 studies, toxics concentrations in many Seattle neighborhoods are heavily impacted by woodsmoke from residential indoor burning and diesel particulate matter. A number of methods are available to determine potential risks from indoor burning, and the current monitoring study includes additional monitoring for woodsmoke related toxic chemicals (PAHs). As described above, these data will not be available until June 2002, and we have elected to use estimates from the PMF modeling exercise performed by Maykut et. al. (2001)[29] to estimate both woodsmoke and diesel particulate matter at Beacon Hill. The annual average woodsmoke and diesel particulate matter concentrations for the Beacon Hill site are multiplied by a woodsmoke and diesel particulate matter unit risk factor, respectively, to estimate the potential cancer risk. These estimates are added to the overall estimated cancer risks from the other monitored air toxics to compare the potential impacts. This information is shown in Figure 3-9 below.

As indicated on Figure 3-9, diesel particulate matter is the greatest contributor to cancer risk estimates at both Beacon Hill and Georgetown. It is possible that the Georgetown risk estimates could be higher since the diesel particulate matter value for this site is assumed to be the same as the concentration estimated at the Beacon Hill site. As noted above, the Georgetown monitor is located in an industrial area that could be heavily impacted by heavy mobile sources such as diesel trucks, and is located in the Duwamish Valley where pollutants may accumulate during inversions.

As indicated on the figure, woodsmoke can contribute significantly to the overall estimated cancer risks from ambient pollution. However, there are a number of areas, such as Lake Forest Park, Puyallup, and Marysville, where the woodsmoke concentrations are significantly higher than those measured at Beacon Hill. For example, annual average woodsmoke concentrations range from 11 to 12 µg/m3 in these areas (see Table 3-2). If concentrations are similar to those listed, potential cancer risks from

[29] Maykut et al. 2001.

woodsmoke could be three times those calculated at Beacon Hill. Even though approximations of risk at these sites could not be estimated, data from other sites suggest that woodsmoke is one of the most significant sources of toxics in the area.

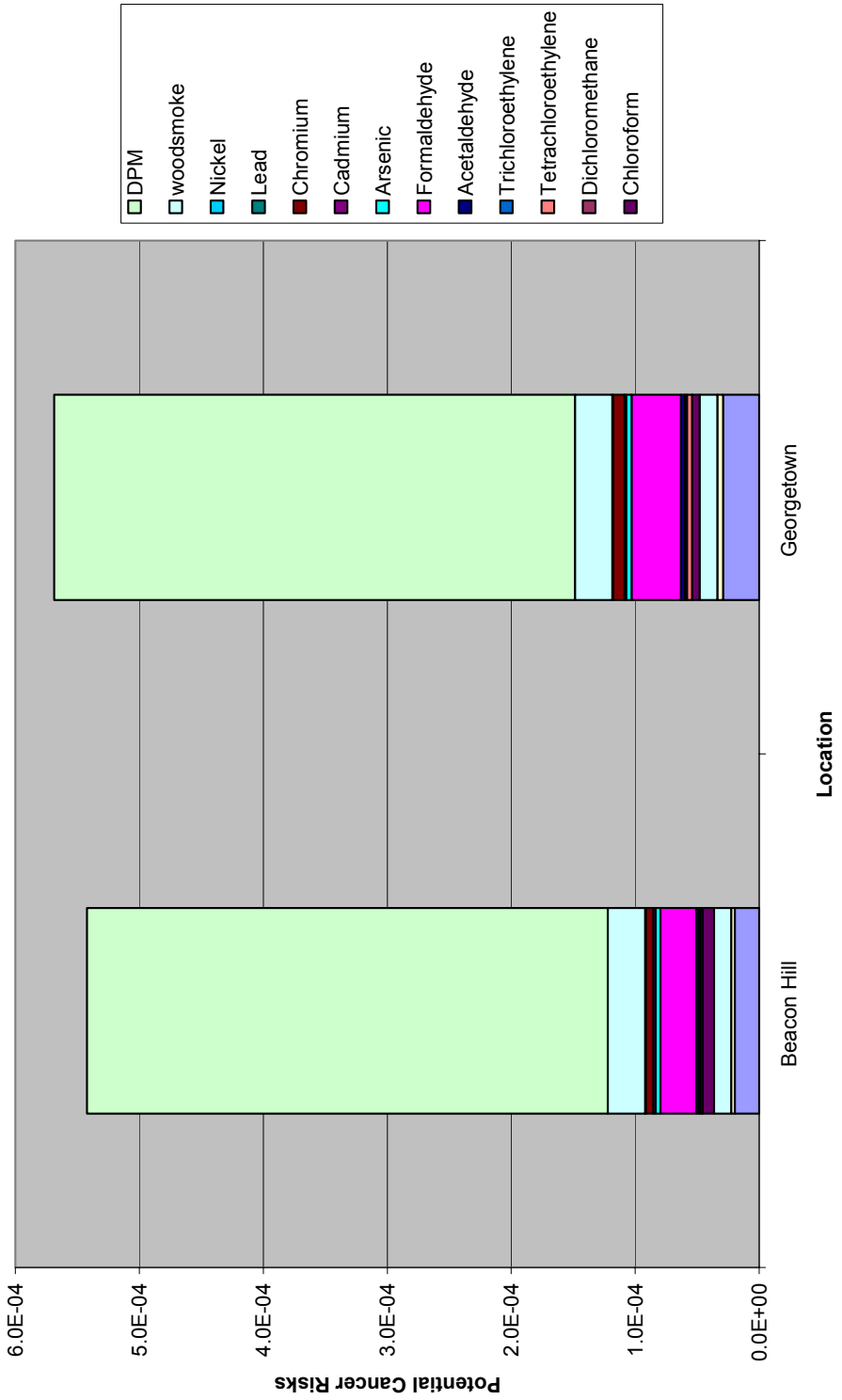


Figure 3-9: Cumulative Cancer Risk Estimates at Beacon Hill and Georgetown

Potential Non-Cancer Effects

Monitored concentrations of toxics were also evaluated for potential non-cancer health effects such as upper respiratory irritation, blood and bone marrow effects, and central nervous system effects. For this exercise, annual average values were compared to RfCs through ratios referred to as hazard indices. The hazard index is a very simple method with which to compare potential exposure concentrations with health-based guidelines (see Chapter 2 for a discussion of the reference concentrations used in this analysis). The hazard indices for the annual averages (the annual average of the Seattle/King County area) are shown in Table 3-4 and Figure 3-10 below.

The annual average was chosen rather than an upper-bound percentile on a 24-hour concentration because upper-bound percentiles were not available for the monitored results. In addition, the annual average may be more representative of potential exposures over the lifetime of the exposed individuals, but it is not a conservative or health protective assumption. The average value is also slightly higher than the median ambient concentration that was used in the USEPA NATA non-cancer evaluation.

All monitored chemicals that had RfCs were evaluated except for woodsmoke. Although it is clear that woodsmoke has serious health effects associated with it, none of the regulatory agencies have developed an RfC for woodsmoke. Both CalEPA and USEPA have derived RfCs for diesel particulate matter, so it is included here.

Table 3-4: Hazard Indices for Monitored Air Toxics

Chemical	Hazard Index for Monitored Chemicals
Benzene	0.02
1,3-Butadiene	0.01
Carbon Tetrachloride	0.04
Chloroform	0.00
Dichloromethane	0.00
Tetrachloroethylene	0.00
Trichloroethylene	0.00
Acetaldehyde	0.14
Formaldehyde	0.15
Arsenic	0.02
Cadmium	0.00
Chromium	0.01
Lead	0.00
Manganese	0.05
Nickel	0.01
Hazard Quotient (sum of the hazard indices)	**0.45**

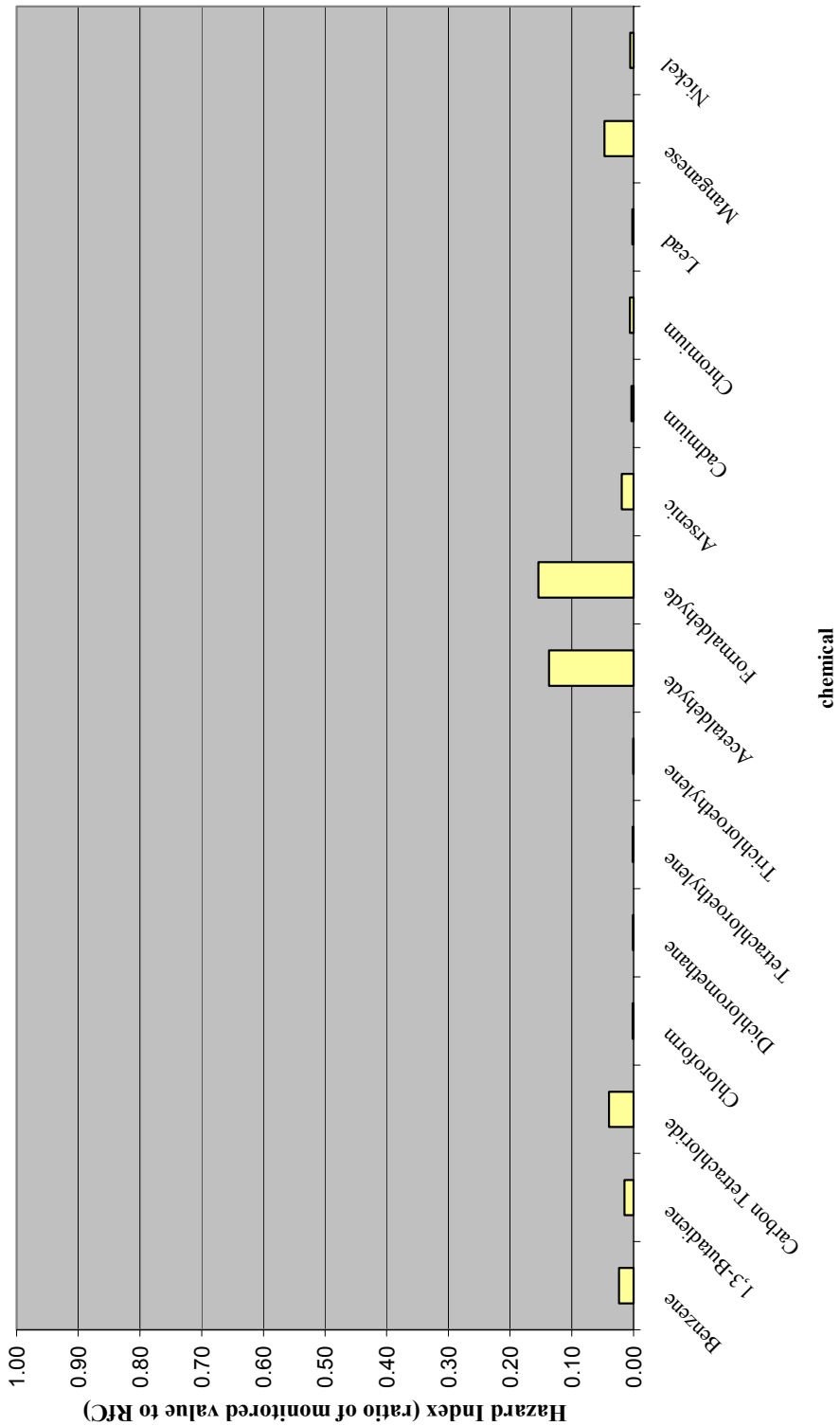


Figure 3-10: Hazard Index for Monitoring Results (average for 6 sites in Seattle/King county)

As shown above, none of the monitored concentrations exceeds or even approaches a hazard index of one. Because some chemicals have the same or similar target organ, some hazard indices can be added together. However, even if all chemicals had the same target organ, the resulting hazard index, referred to as a hazard quotient, is approximately 0.45. This value suggests that potential non-cancer health effects associated with the chemicals monitored in this study alone are not likely to result in significant non-cancer health impacts.

However, these results need to be viewed with caution. The particle-related combustion mixtures, woodsmoke and diesel particulate matter, add a significant amount of PM2.5 into the ambient air. The health effects associated with fine particles include with a wide range of respiratory health effects in humans, and are extensively evaluated elsewhere.[30] In addition, one toxic pollutant, acrolien, could contribute significantly to non-cancer health impacts but was not measured in the monitoring study. More information on non-cancer health impacts are presented in the following chapter on the modeled chemicals.

[30] USEPA ***Third External Review Draft of Air Quality Criteria for Particulate Matter***. EPA/600/P-99/002ac. Office of Research and Development. Research Triangle Park, NC. April 2002.

Chapter 4: Air Toxics Modeling: USEPA NATA Project

Overview

Risks from airborne toxics can also be evaluated and ranked using emission estimates for the primary source categories and dispersion models. This type of assessment has recently been completed by the U.S. Environmental Protection Agency (U.S. Environmental Protection Agency) in their nationwide project entitled the National-scale Air Toxics Assessment (NATA). The results from this analysis for King County were used to evaluate and rank potential risks from airborne toxics. These results were compared to those measured in the monitoring study (see Chapter 3).

The NATA project consists of four phases. In Phase I, USEPA calculates emissions for mobile, area, and point source categories for a total of 33 pollutants and diesel particulate matter. USEPA then uses an air dispersion model (ASPEN, which is explained in more detail later) to predict ambient concentrations for these pollutants (Phase II). In Phase III, USEPA then uses the predicted ambient concentrations to predict human exposure concentrations (the HAPEM4 model, which is also described later in this section). This model accounts for individual movements through various micro-environments such as traveling in a vehicle on the highway, living nearer to significant point sources, and remaining indoors for a portion of each day. Finally, in Phase IV, these human exposure concentrations are used to calculate potential cancer risks and non-cancer risks. Details on the methods and results for each of these phases can be found in the USEPA technical support documents for the NATA project. General information on the four phases is briefly described below.

Phase I: Emission Inventories

In Phase I, USEPA calculates emissions estimates for each of the 34 pollutants from mobile, area and point sources. The 34 pollutants are a subset of the 189 hazardous air pollutants (HAPs) listed in the federal Clean Air Act. This subset was determined by an

emission inventory ranking developed by USEPA.[31] This ranking identified 33 chemicals that were expected to contribute the highest risks from airborne toxics. USEPA also added diesel particulate matter to complete the list of 34. Dioxins were originally included in the 34 chemicals, but USEPA recently removed this suite of chemicals. Although coke oven emissions are included in the NATA project, King County does not have any source of this pollutant. Therefore, it is removed from the list. The final list of 32 chemicals used in this analysis is presented in Table 4-1 below:

[31] USEPA, July 1999.

Table 4-1: Pollutants Included in the NATA Project

Pollutant	CAS Number
Acetaldehyde	75070
Acrolein	107028
Acrylonitrile	107131
Arsenic compounds	NA
Benzene	71432
Beryllium compounds	NA
1,3 Butadiene	106990
Cadmium compounds	NA
Carbon Tetrachloride	56235
Chloroform	67663
Chromium compounds	NA
1,3 Dichloropropene	542756
Diesel particulate matter (DPM)	NA
Ethylene dibromide (1,2 dibromoethane)	106934
Ethylene dichloride (1,2 dichloroethane)	107062
Ethylene Oxide	75218
Formaldehyde	50000
Hexachlorobenzene	118741
Hydrazine, hydrazine sulfate	302012
Lead compounds	NA
Manganese	NA
Mercury compounds	NA
Methylene chloride	75092
Nickel compounds	NA
Polychlorinated biphenyls (PCBs)	1336363
Polycyclic organic matter (POM)	NA
Propylene dichloride (1,2 dichloropropane)	78875
Quinoline	91225
1,1,2,2, tetrachloroethane	79345
Tetrachloroethylene (perchloroethylene)	127184
Trichloroethylene (TCE)	79016
Vinyl chloride	75014

In Phase I, USEPA used emissions estimates from 1996 inventory reporting and estimates, as listed on the USEPA database referred to as the National Toxic Inventory (NTI). USEPA also used information from the National Emission Trends inventory to supplement information for chemicals that may be formed from pre-cursors in the atmosphere.

In addition, USEPA took several steps to perform quality assurance on the emissions estimates. For example, USEPA filled in missing or erroneous information for sources that were missing or poorly reported in the NTI. Emissions estimates in NTI are primarily obtained from state and local inventories, USEPA Maximum Achievable Control Technology information, the Toxics Release Inventory and emissions from USEPA's Office of Transportation and Air Quality. USEPA also requested that individual state and local agencies review emission estimates calculated for the NATA project and submit changes to USEPA before the dispersion-modeling phase was conducted.

USEPA also grouped similar compounds together for more complete evaluation. For example, some chemicals such as various lead or chromium compounds are evaluated together as groups of compounds. In addition, these groups are subdivided according to particle size for more accurate dispersion modeling. Finally, pollutants are assigned to reactivity classes to account for atmospheric decay.

Source Categories

Total pollutant emissions are calculated from point sources, mobile sources, and area sources. Major or point sources are large stationary sources that emit more than 10 tons per year of any HAP or a cumulative total of 25 tons per year of any combination of the 189 HAPs. Area sources are smaller stationary sources. Some smaller facilities do submit emissions inventory reports but the majority of the calculations for area sources are estimated as a ratio to countywide population estimates. USEPA also included other types of area sources such as forest fires and prescribed burning. On-road mobile sources include cars, trucks, buses, etc., while off-road mobile sources include all remaining mobile sources such as trains, boats, lawnmowers, construction vehicles, and aircraft.

Phase II: Predicting Ambient Air Concentrations

ASPEN Model

After the emissions estimates are calculated, the information is entered into the USEPA model referred to as the Assessment System for Population Exposure Nationwide

(ASPEN) air model. This model essentially combines a Gaussian dispersion model with climatological information for each census tract across the United States. ASPEN considers the rate of release of each chemical, the location of the release, the release height, winds speed and direction from the nearest meteorological station, weather (e.g., wet and dry deposition), pollutant decay, atmospheric transformation, and general settling.

Background Concentrations

USEPA also added a "background concentration" for 13 of the 33 pollutants. These concentrations account for toxics that are due to natural sources (e.g., windblown soils, volcanic eruptions, etc.), sources not included in the emissions estimates, and long-range transport. The values included in the analysis as background are typically monitored concentrations in areas that are not heavily impacted by other sources. USEPA refers to these remote areas as "clean air locations." If background concentrations were not available in the literature, the concentrations were assumed to be zero except for diesel particulate matter. For this pollutant, background concentrations were adopted from modeling exercises. This is described more fully in Appendix F of the NATA Science Advisory report.[32]

Phase III: Predicting Human Exposures

HAPEM4 Model

Predicted ambient concentrations are then entered into another model to account for personal exposures and variation among the population in terms of activities. The model used by USEPA is referred to as the Hazardous Air Pollutant Exposure Model, version 4 (HAPEM4). This model evaluates the long-term inhalation exposures by tracking individuals who are considered to be representative of various demographic groups as they move through different locations. These smaller locations are referred to as "micro-environments" and include spaces such as inside a car or bus while traveling along a roadway, inside a home located next to a major source or a number of area sources, or inside a public garage. These are a few of the micro-environments included in the

[32] USEPA, 2001. Appendix F.

HAPEM4 model. A complete list and more detailed descriptions of each micro-environment are include in the technical support documentation for this model.[33]

The model predicts concentrations in these micro-environments and calculates a time-weighted average depending on the amount of time spent in each micro-location. A total of 37 micro-environments were used in predicting the human exposure concentrations for the NATA project.

The HAPEM4 model includes both population activity pattern data and commuting pattern data. Activity patterns include the amount of time people spend at home, work, or in an automobile along with the activities during that time (e.g., sleeping, eating, etc.). HAPEM4 estimates exposures by activity pattern for various demographic groups as defined by age, gender, or race, etc. The commuting pattern data is based on a 1990 U.S. census tract database that reports the number of individuals who work within the census tract where they live.[34]

Pollutant concentrations within each micro-environment are estimated using ambient concentrations multiplied by a penetration factor which is a ratio of the indoor to the outdoor concentration. A time-weighted average exposure concentration can be predicted using these factors and the ambient concentration data for a specified amount of time.

In calculating an annual average estimate for the NATA project, USEPA selected 40 demographic groups based on different combinations of characteristics (e.g., age, race, gender). For each of these groups, 365 activity patterns were randomly selected. The amount of time spent in each micro-environment (for eight separate time blocks for a 24-hour day) for each demographic group was then averaged for the entire set of 365. This process was repeated 100 times for each demographic group so that 100 annual activity

[33] USEPA. ***Development of Microenvironmental Factors for the HAPEM4 in Support of the National-scale Air Toxics Assessment (NATA).*** External Review Draft. Prepared for the Office of Air Quality Planning and Standards. Prepared by ICF Consulting and TRJ Environmental Inc., Research Triangle Park, NC, May 8, 2000.

patterns were available for each of the 40 groups. For each census tract, 30 of these 100 patterns were randomly selected to represent a typical annual time allocation in each micro-environment for demographic groups in that tract. USEPA notes that this process leads to an annual activity that reflects the average exposure in each group, as opposed to highly sensitive or highly exposed individuals.

Potential Cancer Risks

Potential cancer risk estimates are presented in tables 4-2, 4-3 and 4-4 below. As illustrated in Table 4-2 below, the potential cancer risks are similar for the median concentrations from both ASPEN and HAPEM4.[35] The only chemicals that appear to present significantly different cancer risks for ASPEN versus HAPEM4 are tetrachloroethylene or perchloroethylene, and PCBs.

[34] USEPA, January 2001.

[35] Median concentrations were used to compare risk estimates based on ASPEN and HAPEM4 because NATA only provides median values for the HAPEM4 results.

Table 4-2: Comparing Potential Cancer Risk Estimates based on ASPEN and HAPEM4 Exposures

Chemical	King County Risks based on ASPEN (median)	King County Risk based on HAPEM4 (median)
1,1,2,2-TCA	2.2E-09	1.7E-09
1,3-Butadiene	2.2E-06	6.4E-07
1,3-Dichloropropene	3.9E-07	3.2E-07
Acetaldehyde	1.9E-06	1.7E-06
Acrylonitrile	6.7E-09	5.0E-09
Arsenic	4.2E-07	3.3E-07
Benzene	1.8E-05	1.8E-05
Beryllium	2.8E-08	2.2E-08
Cadmium	5.4E-08	4.2E-08
Carbon Tetrachloride	1.3E-05	9.6E-06
Chloroform	1.9E-06	1.6E-06
Chromium	1.5E-05	3.4E-06
Diesel PM	5.0E-04	3.6E-04
Ethylene Dibromide	1.7E-06	1.3E-06
Ethylene Dichloride	1.6E-06	1.4E-06
Ethylene Oxide	1.5E-07	1.2E-07
Formaldehyde	1.5E-05	1.2E-05
Hexachlorobenzene	4.3E-08	3.4E-08
Hydrazine	1.7E-10	1.4E-10
Lead	1.2E-07	1.0E-07
Methylene Chloride	2.3E-07	1.9E-07
Nickel	1.7E-06	3.2E-07
PCBs	**1.7E-06**	**3.1E-08**
Perchloroethylene	**3.8E-08**	**1.4E-06**
7-PAHs	1.1E-06	NA
POM (total)	8.4E-06	5.7E-06
Propylene Dichloride	1.3E-10	9.8E-11
Quinoline	6.7E-10	5.0E-10
Trichloroethylene	1.4E-06	1.2E-06
Vinyl Chloride	1.4E-09	1.1E-09
Total Estimate Cancer Risk	***5.6E-04***	***4.2E-04***

Table 4-3: Potential Cancer Risks based on Annual Average Concentrations Predicted in King County (ASPEN NATA)

Pollutant	Potential Cancer Risk based on Annual Average
Acetaldehyde	1.73E-06
Acrylonitrile	3.54E-09
Arsenic Compounds	4.77E-07
Benzene	1.84E-05
Beryllium Compounds	3.04E-08
1,3-Butadiene	9.42E-07
Cadmium Compounds	5.47E-08
Carbon Tetrachloride	1.32E-05
Chloroform	1.92E-06
Chromium Compounds	6.05E-06
1,3-Dichloropropene	4.18E-07
Diesel particulate matter	6.18E-04[36]
Ethylene Dibromide	1.69E-06
Ethylene Dichloride	1.59E-06
Ethylene Oxide	4.66E-07
Formaldehyde	1.58E-05
Hexachlorobenzene	4.28E-08
Hydrazine	2.96E-10
Lead Compounds	2.22E-07
Methylene Chloride	2.57E-07
Nickel Compounds	4.83E-07
Perchloroethylene	1.90E-06
Polychlorinated Biphenyls	4.27E-08
Polycyclic Organic Matter	5.91E-06
7-PAH	2.86E-07
Propylene Dichloride	7.89E-11
Quinoline	8.00E-10
1,1,2,2-Tetrachloroethane	7.61E-10
Trichloroethylene	1.74E-06
Vinyl Chloride	9.89E-10
Total Estimated Cancer Risks	***6.9E-04***

[36] Calculated using the CalEPA URF.

Table 4-4: Potential Cancer Risks for Puget Sound Clean Air Counties Based on HAPEM4 Exposure Estimates

	King	Kitsap	Pierce	Snohomish
1,1,2,2-TCA	1.7E-09	1.0E-08	2.7E-09	8.0E-09
1,3-Butadiene	6.4E-07	2.8E-07	4.7E-07	4.1E-07
1,3-Dichloropropene	3.2E-07	1.8E-07	2.8E-07	2.4E-07
Acetaldehyde	1.7E-06	1.2E-06	1.5E-06	1.1E-06
Acrylonitrile	5.0E-09	2.5E-08	6.6E-09	1.7E-08
Arsenic	3.3E-07	1.5E-07	2.5E-07	1.5E-07
Benzene	1.8E-05	1.1E-05	1.6E-05	1.4E-05
Beryllium	2.2E-08	1.0E-08	1.8E-08	1.4E-08
Cadmium	4.2E-08	3.3E-08	4.8E-08	3.1E-08
Carbon Tetrachloride	9.6E-06	9.6E-06	9.6E-06	9.6E-06
Chloroform	1.6E-06	1.6E-06	1.6E-06	1.6E-06
Chromium	3.4E-06	5.8E-06	1.5E-06	2.2E-06
Diesel PM	3.6E-04	2.5E-04	3.1E-04	2.6E-04
Ethylene Dibromide	1.3E-06	1.3E-06	1.3E-06	1.3E-06
Ethylene Dichloride	1.4E-06	1.4E-06	1.4E-06	1.4E-06
Ethylene Oxide	1.2E-07	5.0E-08	1.3E-07	5.6E-08
Formaldehyde	1.2E-05	8.6E-06	1.1E-05	8.7E-06
Hexachlorobenzene	3.4E-08	3.4E-08	3.5E-08	3.4E-08
Hydrazine	1.4E-10	1.7E-11	8.4E-11	4.1E-11
Lead	1.0E-07	4.1E-08	3.6E-08	1.6E-08
Methylene Chloride	1.9E-07	1.1E-07	1.4E-07	1.4E-07
Nickel	3.2E-07	4.1E-07	1.1E-07	1.2E-07
PCBS	3.1E-08	3.0E-08	3.0E-08	3.0E-08
Perchloroethylene	1.4E-06	8.9E-07	1.1E-06	1.1E-06
POM (total)	5.7E-06	3.4E-06	4.1E-06	2.3E-06
Propylene Dichloride	9.8E-11	4.2E-10	1.3E-10	3.0E-10
Quinoline	5.0E-10	1.1E-10	2.7E-10	1.7E-10
Trichloroethylene	1.2E-06	3.6E-07	4.9E-07	9.2E-07
Vinyl Chloride	1.1E-09	3.9E-09	1.7E-09	3.0E-09
Total	**4.2E-04**	**3.0E-04**	**3.6E-04**	**3.1E-04**

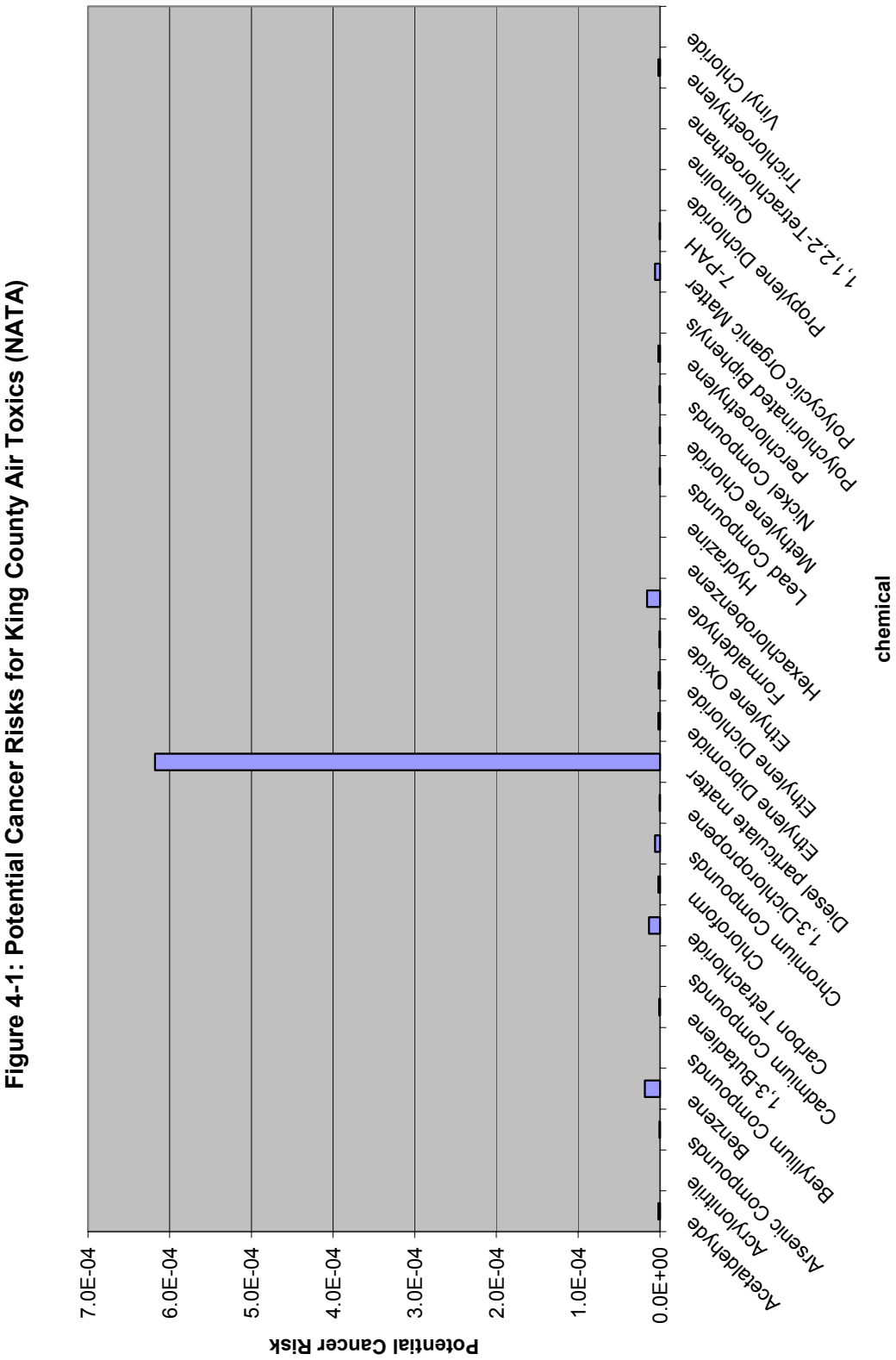


Figure 4-1: Potential Cancer Risks for King County Air Toxics (NATA)

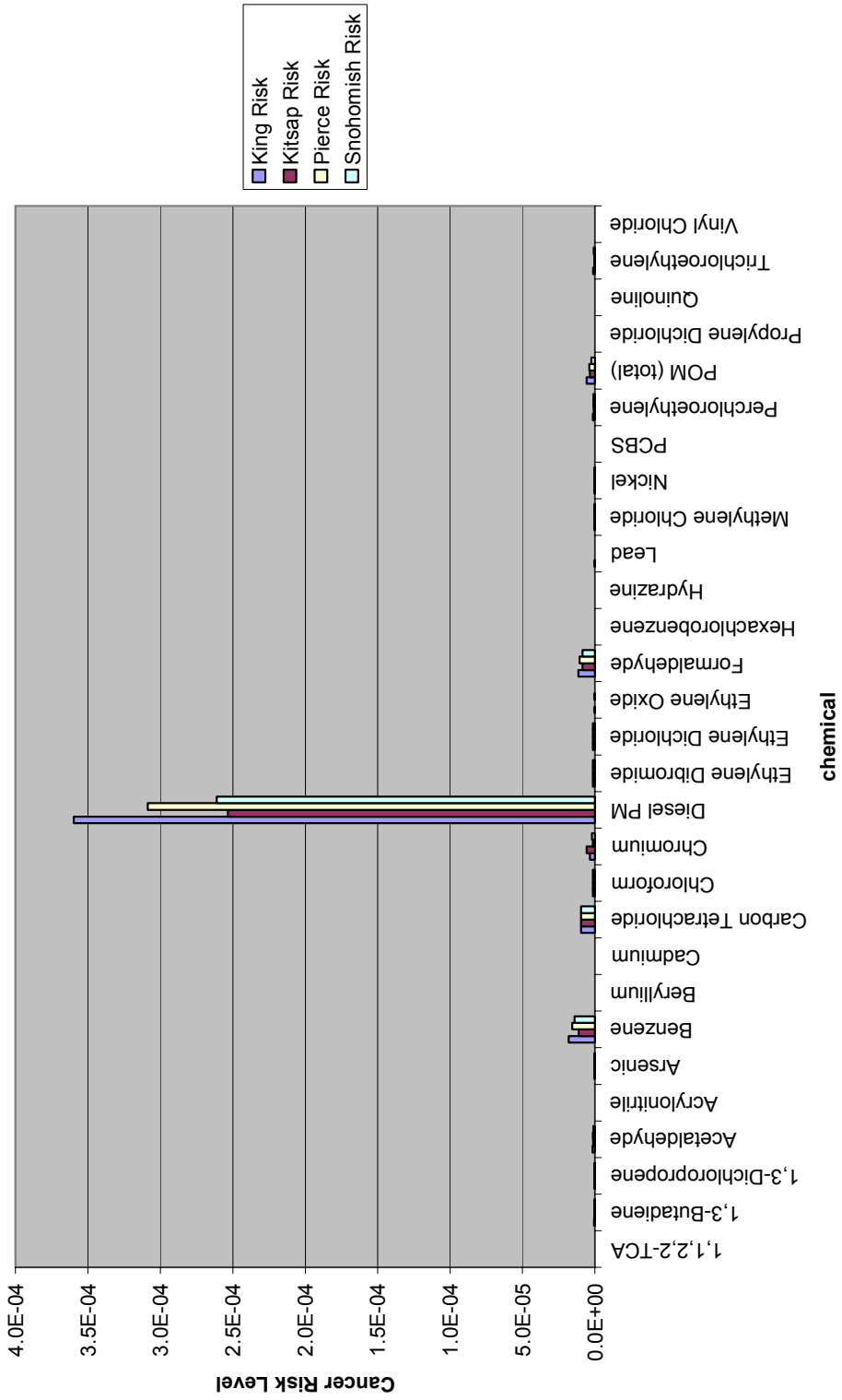


Figure 4-2: Comparing Risks from HAPEM4 for 4 Counties

Figure 4-3: King County Cancer Risks ASPEN NATA

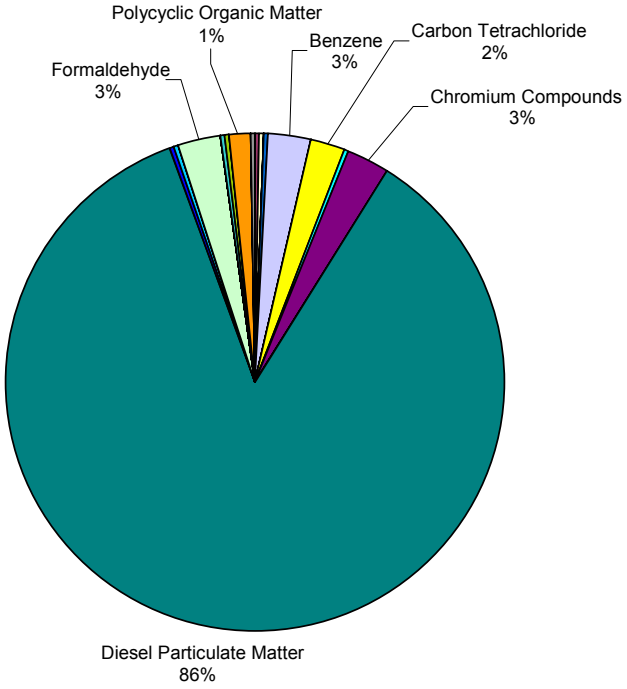
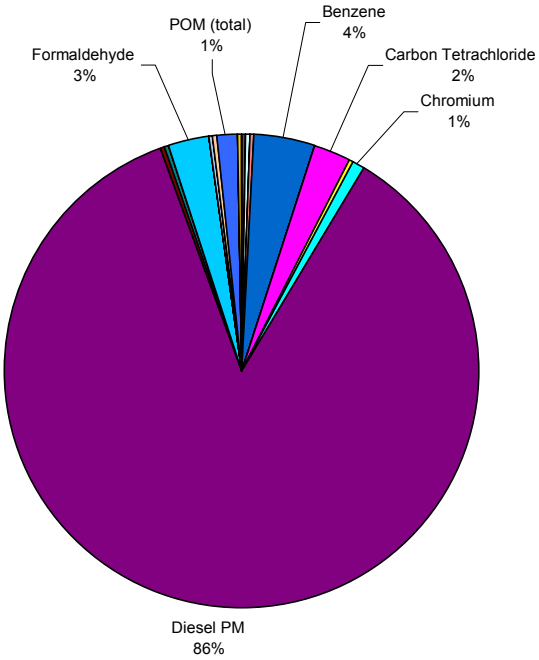


Figure 4-4: Risk Contributions Based on NATA HAPEM4 (median) Estimates



Potential Non-cancer Risks

Table 4-5 presents hazard indices for the range of ambient concentrations predicted by ASPEN. These values are presented not only for the average ambient concentration but for the 75th, 90th, and 95th percentile concentrations as well. The HI associated with the upper percentile concentrations are presented to show the range of potential non-cancer risks. Since we do not have 24-hour-average concentrations that are typically compared to the RfC, a conservative or health protective estimate can be derived using the upper-bound concentration. These concentration ranges are not available for the monitored concentrations or the HAPEM4 (human exposure model) results, so they are not presented in those corresponding sections.

As shown in this table, the hazard indices for most of the 33 chemicals are less than 1.0 for the range of concentrations predicted across King County using the ASPEN model. The only chemical that appears to present a potential non-cancer health risk is acrolein, which has an average hazard index of 6 but could be as high as 12 or higher. The RfC for this chemical is based on irritation effects in the nasal epithelium, although exposure is also associated with irritation of the larynx, trachea, and lungs.[37]

Although this type of analysis indicates that acrolein is the only chemical of those modeled that should be of concern from a non-cancer perspective, it is important to note that the non-cancer health effects associated with particulate matter (e.g., woodsmoke and diesel particulate matter) have not been adequately evaluated using this method. The association between human health effects, such as increased respiratory effects and increased mortality, and ambient exposures to particulate matter are well documented in the literature.[38] As a result, the hazard index for diesel particulate matter should be viewed as only part of the more complex particulate matter threats. Further, it is possible that the non-cancer health effects associated with diesel particulate matter do not have clear thresholds associated with them.

[37] USEPA IRIS file for acrolein. Downloaded February 2002.
[38] USEPA *Air Quality Criteria for Particulate Matter (Second External Review Draft)* EPA 600/P-99/002aB, bB, March 2001

Hazard indices based on the range of exposure concentrations predicted using the HAPEM4 model could not be calculated at this time. USEPA has indicated that they will provide the range of exposure concentrations from the HAPEM4 model results at a later date. We expect to evaluate these concentrations by calculating hazard indices when this information becomes available.

Table 4-5: Hazard Indices for ASPEN ambient estimates in King County

Pollutant	HI for average	HI for 75th	HI for 90th	HI for 95th
Acetaldehyde	0.1	0.1	0.1	0.1
Acrolein	*6.0*	*6.5*	*8.2*	*11.7*
Acrylonitrile	0.0	0.0	0.0	0.0
Arsenic Compounds	0.0	0.0	0.0	0.0
Benzene	0.0	0.0	0.1	0.1
Beryllium Compounds	0.0	0.0	0.0	0.0
1,3-Butadiene	0.0	0.0	0.0	0.0
Cadmium Compounds	0.0	0.0	0.0	0.0
Carbon Tetrachloride	0.0	0.0	0.0	0.0
Chloroform	0.0	0.0	0.0	0.0
Chromium Compounds	0.0	0.0	0.0	0.0
1,3-Dichloropropene	0.0	0.0	0.0	0.0
Ethylene Dibromide	0.0	0.0	0.0	0.0
Ethylene Dichloride	0.0	0.0	0.0	0.0
Ethylene Oxide	0.0	0.0	0.0	0.0
Formaldehyde	0.1	0.1	0.2	0.2
Hexachlorobenzene	0.0	0.0	0.0	0.0
Hydrazine	0.0	0.0	0.0	0.0
Lead Compounds	0.0	0.0	0.0	0.0
Manganese Compounds	0.0	0.1	0.1	0.1
Mercury Compounds	0.0	0.0	0.0	0.0
Methylene Chloride	0.0	0.0	0.0	0.0
Nickel Compounds	0.0	0.0	0.0	0.0
Perchloroethylene	0.0	0.0	0.0	0.0
Polychlorinated Biphenyls	0.0	0.0	0.0	0.0
Polycyclic Organic Matter	0.0	0.0	0.0	0.0
7-PAH	0.0	0.0	0.0	0.0

Propylene Dichloride	0.0	0.0	0.0	0.0
Quinoline	0.0	0.0	0.0	0.0
1,1,2,2-Tetrachloroethane	0.0	0.0	0.0	0.0
Trichloroethylene	0.0	0.0	0.0	0.0
Vinyl Chloride	0.0	0.0	0.0	0.0
Total Hazard Quotient	***6.2***	***6.8***	***8.7***	***12.2***

Chapter 5: Summary and Conclusions

We evaluated cancer and non-cancer risks using three different methods of estimating potential exposures. Each method has associated limitations, strengths, and uncertainties. Regardless of which method is used, the risk estimates are surprisingly consistent across the greater Seattle/King County urban area. They are also similar in the remaining three counties in the Puget Sound jurisdiction (Kitsap, Pierce, and Snohomish). The estimated cancer risks range from a median value of 300 in one million for all 32 HAPEM4 modeled toxics (including diesel particulate matter) in Snohomish County to a high of 690 in one million as an average for 32 modeled ambient concentrations in King County (including diesel particulate matter). The highest risk estimates based on monitored data are 560 in one million, using the full year of data (2000) in Georgetown. This risk estimate includes the 15 air toxics, plus woodsmoke and diesel particulate matter. All risk estimates reflect a 70-year exposure period.

The air toxics that contribute most to the cancer risks are also very consistent across the different methods of analysis. The top toxics for all three methods include diesel particulate matter, benzene, formaldehyde, carbon tetrachloride, and chromium compounds. Woodsmoke contributed significantly in the risk estimates based on the monitored data. In addition, the percent contribution of the top air toxics is also very similar across the different methods of analysis. For example, at Beacon Hill, diesel particulate matter accounts for over 70% of the potential cancer risks, with woodsmoke contributing approximately 6%. The remaining 23% is due to air toxics primarily related to mobile sources (see Figure 5-1). The King County results from the ASPEN model estimate diesel particulate matter at 86%, with other mobile-source-related chemicals at about 8%, and stationary-source-related chemicals at about 6% (see Figure 5-2). Similarly, the HAPEM4 results indicate a diesel particulate matter contribution of 86%, with other mobile source related chemicals at 7%, and stationary sources at about 4% (see Figure 5-3).

Figure 5-1: Contributions to Estimated Cancer Risks at Beacon Hill (2000 monitored data)

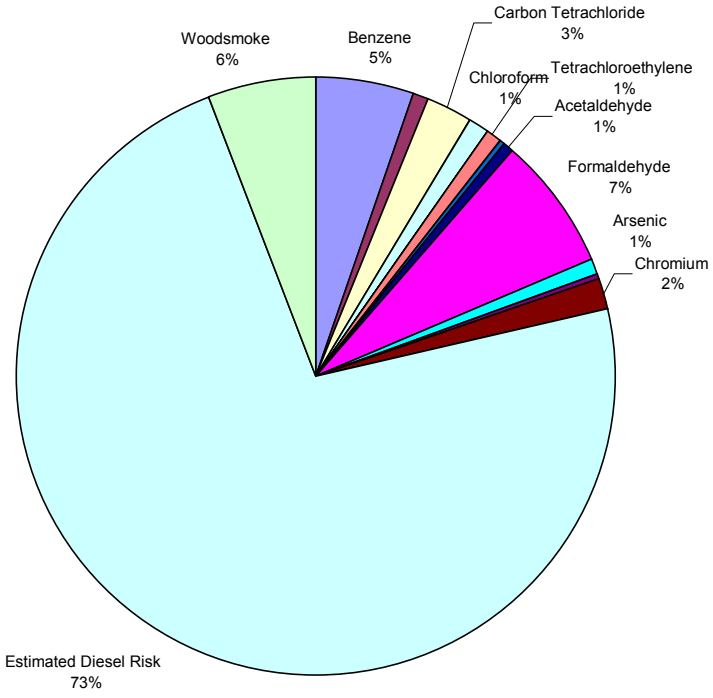


Figure 5-2: King County Cancer Risks ASPEN NATA

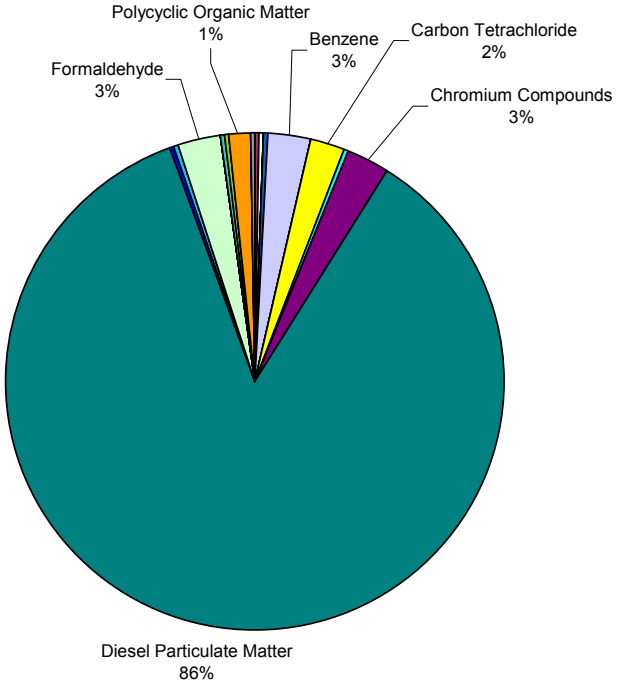
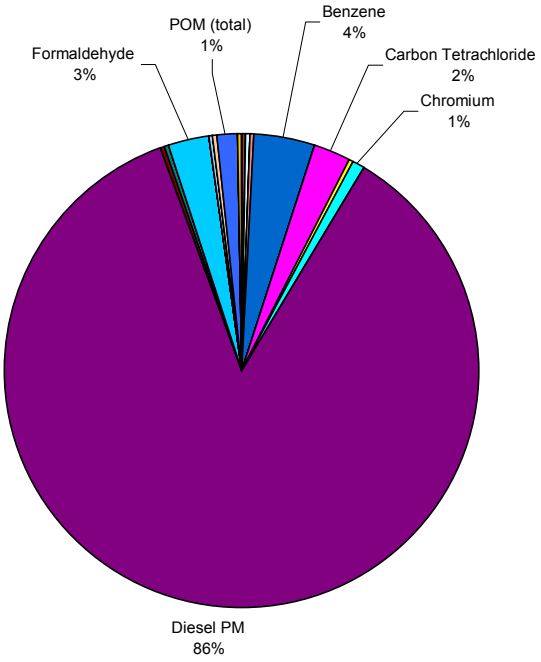


Figure 5-3 : Risk Contributions Based on NATA HAPEM4 (median) Estimates



Cancer risk estimates, even when human and pollutant movement are considered, are also similar among the different methods of calculating exposures concentrations. For example, cancer risk estimates for King County alone range from approximately 400 to 700 in a million based on HAPEM4 and ASPEN modeled data, respectively. Similarly, the cancer risk estimates for the monitored data are approximately 550 in a million for the greater Seattle/King County area.

The most complete set of monitored data comes from the Beacon Hill site for the 2000 calendar year. The data from this site indicate an average cumulative cancer risk of approximately 550 in one million over a 70-year exposure period. This site includes levels for woodsmoke, diesel particulate matter, and 14 other toxic chemicals that are typical of an urban residential area. Results from the 2001 Seattle Air Toxics Monitoring study suggest that other areas throughout Seattle and King County are similar to the Beacon Hill site (see Figure 3-3).

Uncertainties

Although the modeled concentrations provide the highest cancer risk estimates, these values are likely to underestimate the risks from air toxics when compared with the monitor-based risk estimates. It is not clear why the ASPEN model underestimates ambient concentrations in the King County area, although it is possible that the modeling reflects ambient concentrations across the entire county, which includes less impacted areas. However, even measurements taken at the more rural location, Lake Sammamish, are quite similar to concentrations measured in other areas across the greater Seattle area. It is possible that the emissions inventory does not include all sources or underestimates emissions. Another possibility is that the model does not adequately consider production of HAPs from atmospheric transformation reactions. However, the NATA ambient concentrations (ASPEN) result in larger cancer risk estimates than the monitored estimates because they include a larger number of air toxics than the monitoring studies. This suggests that if the list of air toxics were more comprehensive, the overall estimated cancer risks could increase, although it is difficult to say by how much.

In addition underestimating ambient concentrations, model-based risk estimates appear to greatly underestimate the cancer risks from woodsmoke. In the modeling analysis, woodsmoke is included in the general area source category for the 7 PAHs. Since PAHs do not appear as a significant contributor in either of the model-based risk results (i.e., either the HAPEM4 or ASPEN), it follows that woodsmoke does not present a significant risk when evaluated this way. When evaluated using the entire mixture and a unit risk factor for woodsmoke, the cancer risks from woodsmoke increase significantly. In addition, woodsmoke concentrations for areas more heavily impacted by indoor burning could be significantly greater than those measured in the Beacon Hill. Previous woodsmoke estimates for Puyallup and Lake Forest Park could increase the average cancer risks by another 100 in a million.[39]

Because the risk values are based on annual average or median exposure concentrations which are combined with conservative toxicity estimates, they are expected to be reasonable high-end risk estimates but not maximum risk estimates. For some chemicals, the values may underestimate potential cancer risks for some individuals. The concentrations used in the risk calculations are county-wide averages that may not reflect local hotspots. For example, individuals who spend more of their time near large point sources may experience higher risks due to those emissions.

Alternatively, much of the air monitoring and human behavioral information suggests that potential cancer risks may not vary dramatically across the county. For example, the monitoring results suggest that average ambient concentrations for a variety of toxics do not appear to vary significantly among different areas of the county, although daily exposures can vary dramatically.

Finally, the results from the HAPEM4 model suggest that behavior patterns and micro-environment locations may reduce exposures (from ambient concentrations), on average, for most chemicals. For example, risk estimates based on the human exposure modeling

[39] Multiply the woodsmoke URF of 1E-05/µg/m3 by the Puyallup or Lake Forest woodsmoke estimate of 11 µg/m3 to obtain 100 in one million.

(HAPEM4) are approximately 25% less than those estimated using the ambient modeling data, although this still suggests that average cancer risks in King County are at least as high as 520 in a million.[40] However, it is important to note that there are some significant limitations with the HAPEM4 model. There is not enough information on many chemicals to inform model results regarding the potential exposures in micro-environments. For example, while most chemicals do not differ in their exposure concentrations (e.g., HAPEM4 results) from their ambient concentrations, it is interesting to note that the HAPEM4 and ASPEN estimates for tetrachloroethylene vary by almost two orders of magnitude. It is possible that there is more indoor monitoring data available for this chemical to refine the model estimates, but not for other chemicals. However, it is also possible that the HAPEM4 results reflect the fact that the majority of the chemicals have relatively long atmospheric lifetimes. This suggests that indoor concentrations will be similar to outdoor concentrations over time, and that these chemicals move more easily among certain micro-environments.[41] This could account for the similarity in risk estimates between the ASPEN and HAPEM4 concentrations.

Finally, the cancer risk estimates for diesel particulate matter also have some uncertainties associated with them. Although USEPA has not recommended a final unit risk factor for evaluating potential cancer risks associated with environmental exposures to diesel particulate matter, they state strongly that diesel particulate matter is a probable human carcinogen. In addition, USEPA encourages states to consider further the possible range of potential cancer risks associated with those levels predicted using the NATA results. In the NATA document, USEPA states[42]

> *Even the lower end of the risk range (presented in the risk perspectives section of the Diesel Exhaust HAD) is above the level that has historically warranted regulatory concern at USEPA for air toxics. The Agency believes that aresa of the U.S. that have relatively higher annual exposure levels for diesel exhaust, certainly those counties and States with annual exposure levels above 2 micrograms per cubic meter, should consider the scientific judgements that the Agency has made in the risk perspectives section of the HAD while considering*

[40] Multiply the ambient model-based risk results of 690 in a million by 75% to obtain 517 in a million.
[41] USEPA, 2000.
[42] USEPA 2001 pg 102.

> *the important limitations in their efforts to compare air toxics risks and set priorities for their programs. At the higher exposure levels found in a number of urban areas in NATA, there is an overlap between what the occupational levels were in the epidemiological studies that EPA considered and the environmentally equivalent exposures.*

Overall, this information suggests that ambient air toxics could contribute significantly to cancer and non-cancer risks in the Puget Sound region. It is possible that this risk is underestimated because 1) not all air toxics are considered in this analysis, and 2) many chemicals have been shown to accumulate in indoor micro-environments, which could increase exposure. Alternatively, risk may be overestimated by assuming that the concentration at the monitor accurately reflects lifetime exposure to ambient pollutants. It is important to note that this analysis does not evaluate indoor sources of air pollution (i.e., from paints, home furnishings, cleaning products, building materials and other indoor sources). Uncertainties in the toxicity information could also serve to over or underestimate potential risk estimates.

The information presented in this report uses screening risk estimates as a tool to focus agency attention on those compounds and mixtures that are likely to present the greatest risk of cancer and some non-cancer effects. Consistency among concentrations measured for a limited number of compounds and modeled ambient concentrations, as well as those modeled concentrations that incorporate more exposure data, gives credibility to this ranking effort. Woodsmoke and diesel particulate matter rank high in potential contributions to cancer and non-cancer risk, higher than other air toxics measured in this study. In addition, volatile organics associated with mobile sources such as benzene, formaldehyde, and 1,3 butadiene contribute significantly to the potential cancer risks from air toxics. Diesel particulate matter, benzene, 1,3 butadiene, and formaldehyde are all classified as class A or B carcinogens under the USEPA cancer rating system. This indicates that USEPA is relatively confident that these chemicals probably cause cancer in humans. These chemicals should have high priority during development of an air toxics reduction program for the Puget Sound area. Finally, acrolein appears to present a potential non-cancer risk as well. As stated earlier, the non-cancer health effects associated with the particulate-matter combustion mixtures (e.g., woodsmoke and diesel

particulate matter) are not adequately evaluated here, and are extensively evaluated in other analyses.

In addition, these analyses suggest that the ambient concentration estimates from mobile sources predict with reasonable accuracy, and that they can be used in the absence of more accurate monitoring data, particularly in urban areas. This conclusion may not apply to model results for more rural areas, particularly if outdoor or agricultural burning could contribute substantially to ambient PM or air toxics concentrations. We also recommend additional review of the HAPEM4 results in future NATA analyses, and further research for exposure models that may more accurately predict potential exposures to air toxics.